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May 29, 1992

Homicide Surveillance, 1979–1988

Influenza – United States, 1989–90 and 1990–91 Seasons

Laboratory-Based Surveillance for Rotavirus – United States, January 1989–May 1991

Chancroid – United States, 1981–1990: Evidence for Underreporting of Cases

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Abbreviations

NCCDPHP	National Center for Chronic Disease Prevention and Health Promotion
NCEHIC	National Center for Environmental Health and Injury Control
NCID	National Center for Infectious Diseases
CIO	Centers/Institute/Offices
NCPS	National Center for Prevention Services
EPO	Epidemiology Program Office
NIOSH	National Institute for Occupational Safety and Health

Homicide Surveillance – United States, 1979–1988

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Summary

From 1979 through 1988, 217,578 homicides occurred in the United States, an average of >21,000 per year. Homicide rates during this 10-year period were about 1.5 times higher than the rates during the 1950s. The national homicide rate of 10.7/100,000 in 1980 was the highest ever recorded. Homicide occurs disproportionately among young adults. Among the 15- to 34-year age group, homicide is the fourth most common cause of death among white females, the third most common cause among white males, and the most common cause among both black females and black males. In 1988, nearly two-thirds (61%) of homicide victims were killed with a firearm, 75% of these with a handgun. More than half (52%) of homicide victims were killed by a family member or acquaintance, and about one-third (35%) of homicides stemmed from a conflict not associated with another felony. The homicide mortality rate among young black males 15–24 years of age has risen 54% since 1985. Ninety-nine percent of the increase was accounted for by homicides in which the victim was killed with a firearm. The surveillance data summarized in this report should assist public health practitioners, researchers, and policymakers in addressing this important public health problem.

INTRODUCTION

Violent and abusive behavior exacts a large toll on the physical and mental health of Americans. Homicide, the fatal outcome of that behavior, is increasingly recognized as an important public health problem in the United States. The national objectives for health promotion and disease prevention call for a 15% reduction in the rate of homicide by the year 2000 (*1*).

This is the third homicide surveillance report published by CDC. The first provided detailed information about homicide mortality in general (*2*). The second focused on high-risk racial and ethnic groups (*3*). This homicide surveillance report focuses on the 10-year period 1979–1988, the most recent years for which complete mortality data have been released. The report provides a written summary and tabular details of homicide in the United States for specific age, race (black, white), and gender groups. Data for black males, white males, black females, and white females are displayed separately, so that patterns unique to each of these four groups can be examined. Hispanic-specific rates are not provided because they are not yet available for all states.

These surveillance reports are intended to assist public health practitioners, researchers, and policymakers in their efforts to become familiar with and address this increasingly important public health problem.

DATA SOURCES

Homicide is defined as death resulting from injuries purposefully inflicted by another person, including deaths caused by law-enforcement officers or legal execution. The information on homicide presented in this report is drawn from two sources: detailed mortality tapes prepared by the National Center for Health Statistics (NCHS), CDC, and the Federal Bureau of Investigation's Supplementary Homicide Report (FBI-SHR).

NCHS Mortality Data

Deaths that resulted from homicide were extracted from the National Mortality Tapes compiled by the NCHS. The information on these tapes is provided by the 50 states and the District of Columbia. The statistics compiled on these tapes include age, race, sex, geographic data, and cause of death, coded according to the *International Classification of Diseases, Ninth Revision* (ICD-9) (*4*). This report includes all deaths during the period 1979–1988 with the underlying cause coded E960-E978. This category includes the following ICD classifications: Homicide and Injury Purposely Inflicted by Other Persons (ICD-9 codes E960-969) and Legal Interventions (includes legal executions and deaths from injuries inflicted by law-enforcement agents in the course of duty) (ICD-9 codes E970-978). Deaths resulting from legal interventions account for 1.3% of the total deaths due to homicide.

In this report, mortality statistics are classified by age, race (black, white), and sex. The white category includes persons classified as white, Mexican, Puerto Rican, Cuban, and all other Caucasians. Other categories (Native American, Asian/Pacific Islander) are included in the totals but have been excluded from the race-specific analyses because the numbers are too small to provide stable rate estimates.

Even though the reporting of Hispanic ethnicity on death certificates has been improving, the data are not yet reported from all states. Therefore, Hispanic-specific analyses have not been conducted. Previously published information indicates that homicide rates among Hispanics are higher than those among non-Hispanic whites (*3,5*).

FBI-SHR Data

The FBI-SHR compiles data that have been submitted voluntarily by >16,000 county, city, and state law-enforcement agencies throughout the United States. In 1988, approximately 98% of the U.S. population was covered by this reporting system (*6*). For the purposes of this data set, homicide is defined as murder and non-negligent manslaughter (the willful killing of one human being by another), including justifiable homicides by private citizens (killing of a felon during the commission of a felony) and killing of a suspected felon by a peace officer in the line of duty (*7*).

In addition to demographic information about the victim similar to that contained in NCHS mortality data, the FBI-SHR also includes demographic information (age, race, sex) on the assailants and information about the weapon used in the homicide, the relationship between victim and assailant, and the circumstances of the homicide (e.g., argument, rape). The data are based on the reports of the investigating law-enforcement officials.

As with analyses done with NCHS data, race-specific analyses based on FBI-SHR data include black and white only. Other races (Native American, Asian/Pacific Islander) are included in the totals but not in the race-specific analyses.

The NCHS U.S. Vital Statistics data routinely report about 10% more homicides per year than FBI-SHR data (Table 1). The difference is presumed to arise from the voluntary nature of the FBI-SHR reporting system. The demographic patterns, however, are quite similar between the two data sources, and inferences drawn from either data set are probably valid for the other (*8*). Most tables in this report display NCHS data because they are more complete. However, information about the offender and the circumstances of the homicide is available only in FBI-SHR data, and the specific type of firearm (e.g., handgun, rifle) is more completely reported in the FBI-SHR than in the NCHS statistics. Analyses pertaining to these variables are based on FBI-SHR data.

Population Data

Population data used to calculate rates for 1979–1988 were based on the 1980 census. Intercensal estimates were performed under contract by Richard Irwin, Demo Detail, Alexandria, VA.

RESULTS

Homicide as a Cause of Death

From 1979 to 1988 there were a total of 217,578 deaths due to homicide in the United States (Tables 2a, 3a, Figure 1). The homicide rate in 1980 of 10.7/100,000 was the highest of the decade and the highest rate ever recorded for the United States. From 1980 through 1985, the rate declined to a 10-year nadir of 8.3/100,000, after which it increased again, ending at 9.0/100,000 in 1988.* In 1988 homicide was the fifth leading cause of years of potential life lost.

Rates of Homicide by Race and Sex (Tables 2d–g, Figures 2,3)

The majority of homicide victims were white, although homicide rates were higher among blacks. From 1979 to 1988, there were 217,578 victims of homicide; 88,162 (40%) were white males, 75,832 (35%) were black males, 30,644 (14%) were white females, 18,274 (8%) were black females, and 4,484 (2%) were classified as "other." Throughout the decade, homicide rates were highest among black males, followed by black females, white males, and white females. Rates for these four race-gender groups declined during the first half of the decade. From 1986 through 1988, however, the rates for white males and white females remained stable or continued to decline, while the rates increased 40% for black males and 20% for black females.

Rates of Homicide by Age, Race, and Sex (Tables 2a,2d–g,4a–e, Figure 4)

Homicide is more common among young adults. From 1979 through 1988, 56% of all homicide victims were 15–34 years of age. Homicide is the second most common cause of death among persons 15–34 years of age, exceeded only by unintentional injuries. In this age group, homicide was the fourth most common cause of death among white females, the third most common cause among white males, and the most common cause among both black females and black males.

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*Provisional rates for 1989 and 1990 are 9.3 and 10.2 per 100,000, indicating that the rate has continued to climb (*9*).

Throughout the 10-year period, homicide rates were highest for 25- to 34-year-old white males, black males, and black females. For some years during that period, homicide was also the leading cause of death for 15- to 24-year-old white females.

The increase in the rate from 1985 to 1988 among black males was largely accounted for by an increase in homicides among black males 15–34 years of age, especially among those 15–24 years of age. For black males 25–34 years of age, the rate increased 15% (from 94.4/100,000 to 108.9/100,000). Among black males 15–24 years of age, the rate increased 54% (from 66.1/100,000 to 101.8/100,000).

Geographic Patterns of Homicide (Table 5, Figure 5)

Crude homicide rates are generally higher among states in the southeast; rates that have been adjusted for age, sex, and race are higher in the west. When data are combined for the years 1986–1988 to achieve greater stability of the observed rates, six of the 10 states with the highest crude rates are in the southeast and two are in the west. For the adjusted rates, only two of the top 10 states are in the southeast and five are in the west.

Homicide Weapons (Table 6, Figures 6–7)

In 1988, 61% of all homicides involved the use of a firearm, and 75% of these were committed with a handgun. The type of weapon involved depended more on the sex than the race of the victim. A lower proportion of female homicide victims was killed with firearms (45%) compared with male homicide victims (67%). The difference was largely due to "other weapons" (i.e., weapons other than guns, knives, or cutting instruments), which accounted for 28% of homicides of females but only 12% of homicides of males.

The proportion of homicide victims killed with a gun was highest for black males (70%), with 55% of all black male victims killed with a handgun. The rate of firearm homicides closely parallels the rate of total homicides, particularly among young black males.

Relationship of Victim to Assailant (Table 7, Figures 8–10)

In 1988, more than half (52%) of homicide victims were killed by family members (14%) or acquaintances (38%). The proportion of females killed by a family member (27%) was considerably higher than the proportion of males killed by a family member (10%).

In 1988, 87% of assailants whose sex was recorded were males. In 63% of homicides, both victim and assailant were male; in 24%, females were killed by males; in 11%, males were killed by females; and in 2%, both victim and assailant were females.

In most homicides, the victim and perpetrator were of the same race. In 1988, 48% of all homicides involved black victims who were killed by blacks, while 43% involved white victims who were killed by whites.

Homicide Circumstances (Table 8)

In 1988, the circumstance most frequently cited by the law-enforcement official was a conflict (35%), which includes brawls under the influence of alcohol, lovers' triangles, children killed by babysitters, disputes over money, and other arguments. Although the circumstances of a large proportion (28%) of homicides were unknown, only 13% were said to have occurred in association with another felony.

Unlike the mutually exclusive characteristics used to categorize the weapon and the assailant (e.g., firearm vs. knife, family member vs. acquaintance), determining the circumstance of a homicide often requires the responsible law-enforcement officer to choose among several possibilities. For example, there may have been a conflict over property or money; the victim, assailant, or both may have been using drugs or alcohol; and the victim, assailant, or both may have been members of a gang. Although all three factors may have contributed to the homicide, the recording officer must select only one category as the single most appropriate circumstance. Appreciable variation among jurisdictions exists and is likely due to different recording practices (*10*). Thus, changes over time in the distribution of homicides by circumstance may be real trends or trends in the recording practices of the various reporting agencies.

DISCUSSION

Homicide is an especially important public health problem among young adults in the United States. Only within the past 10 years, however, have public health workers begun to apply systematically the methods of public health surveillance, epidemiologic research, intervention, and evaluation to homicide and other adverse outcomes of violence.

The data in this surveillance report further characterize the magnitude of the problem. Important trends and risk groups, such as the sharply increasing homicide rates among young black males, are identified. Nevertheless, homicide affects all segments of American society, is a leading cause of death among young persons of all race and gender groups, and is far more common in the United States than in other similarly developed nations (*11*).

The epidemiologic analyses presented in this surveillance report also indicate that homicide—and the violence that leads to homicide—is a multifaceted problem. Homicides of infants, homicides of sex partners, and homicides committed during robberies are but a few of the readily distinguishable types of homicide, each type having its own epidemiologic pattern and unique set of causes. These and other types of homicide need thorough epidemiologic characterization so that more effective and targeted prevention programs can be developed.

Although the various categories of homicide have unique patterns and causes, they have some common elements as well. Firearms, for example, are the most common weapon overall and, with the exception of child homicide, presumably the most common weapon used in each of the different types of homicide. Similarly, although less easily defined and incriminated than firearms, a cultural acceptance of and an inclination toward violent resolution of conflicts also play a part in the increasing rates of homicide (*12*).

While scientific studies of these and other probable causes of homicide continue, the design, implementation, and evaluation of interventions have begun (*13*). Given the multiple types of homicide and the complex web of factors causing each, no single solution to the prevention of violent injuries and deaths can be expected. Multiple interventions in multiple settings will be necessary. Although the redress of certain contributing factors, such as poverty and racism (*14–18*), is likely to require efforts at the national level, many interventions are most likely to succeed if originated, implemented, and controlled at the community level. Some of the possible interventions that can be incorporated into a community program are

mentoring programs, school-based curricula in nonviolent conflict-resolution skills, peer-counseling programs, enforcement or enactment of local drinking and firearm control regulations, special recreational programs for youth, and home visitation programs by nurses for young, poor, single-parent mothers (*19*).

The data in this report show that homicide rates vary among population groups and geographic location, and over time. Although the factors that account for these changes are not always clear, the variations themselves suggest that homicide rates and other manifestations of violence can be modified. Continued surveillance, plus augmented efforts in the areas of epidemiologic research, intervention, and evaluation, should be helpful in reducing the large burden of violence-related injuries and deaths.

References

1. U.S. Department of Health and Human Services. Healthy people 2000: national health promotion and disease prevention objectives. U.S. Public Health Service. DHHS Publication No. (PHS) 91-50212. Washington, DC: 1991, p. 228.
2. CDC. Homicide surveillance report, 1970–1978. Atlanta: Centers for Disease Control, September 1983.
3. CDC. Homicide surveillance: high-risk racial and ethnic groups—blacks and Hispanics, 1970–1983. Atlanta: Centers for Disease Control, November 1986.
4. U.S. Department of Health and Human Services. The international classification of diseases: 9th revision, clinical modification. U.S. Public Health Service. DHHS Publication No. (PHS) 89-1260. March 1989.
5. CDC. Advance report of final mortality statistics, 1989. Monthly vital statistics report; vol. 40, no. 8 (suppl. 2). Hyattsville, MD: Public Health Service, 1992, p. 8.
6. Federal Bureau of Investigation. Crime in the United States: uniform crime reports, 1988. Washington, DC: U.S. Department of Justice, 1988.
7. Federal Bureau of Investigation. Uniform crime reporting handbook. Washington, DC: U.S. Department of Justice, 1984.
8. Rokaw WM, Mercy JA, Smith JC. Comparing death certificate data with FBI crime reporting statistics on U.S. homicides. Public Health Reports 1990;105:447–55.
9. CDC. Annual summary of births, marriages, divorces, and deaths; United States, 1990. Monthly vital statistics report; vol. 39, no. 13. Hyattsville, MD: Public Health Service, 1991.
10. Maxfield MG. Circumstances in supplemental homicide reports: variety and validity. Criminology 1989;27:671–95.
11. Fingerhut LA, Kleinman JC. International and interstate comparisons of homicide among young males. JAMA 1990;263:3292–5.
12. National Committee for Injury Prevention and Control. Injury prevention: meeting the challenge. Am J Prev Med 1989;5(suppl):192–203.
13. Proceedings of a panel discussion. Lessons learned—the community experience. Public Health Reports 1991;106:237–41.
14. Rosenberg ML, O'Carroll PW, Powell KE. Let's be clear: violence is a public health problem. JAMA 1992;267:3071–2.
15. Blau JR, Blau PM. The cost of inequality: metropolitan structure and violent crime. Am Sociol Rev 1982;47:114–29.
16. Messner SF. Regional differences in the economic correlates of the urban homicide rate: some evidence on the importance of the cultural context. Criminology 1982;21:477–88.
17. Messner SF. Poverty, inequality, and the urban homicide rate: some unexpected findings. Criminology 1982;20:103–14.
18. Silberman CE. Criminal violence, criminal justice. New York: Vintage, 1978.
19. CDC. The prevention of youth violence: a framework for community action. Atlanta (in press).

TABLE 1. Number of homicides and homicide rates,* by race of victim, year of death, and source of data† — United States, 1979–1988§

	White				Black				Total ¶			
	NCHS		FBI		NCHS		FBI		NCHS		FBI	
Year	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
1979	12,334	6.4	11,518	6.0	9,812	37.6	9,384	36.0	22,550	10.0	21,417	9.5
1980	13,558	7.0	12,055	6.2	10,283	38.4	9,759	36.4	24,278	10.7	22,786	10.0
1981	13,066	6.6	11,226	5.7	10,137	37.3	9,252	34.0	23,646	10.3	20,931	9.1
1982	12,439	6.3	11,159	5.6	9,473	34.3	8,658	31.3	22,358	9.6	20,288	8.8
1983	11,235	5.6	10,604	5.3	8,494	30.3	8,316	29.6	20,191	8.6	19,426	8.3
1984	11,127	5.5	9,999	5.0	8,240	29.0	7,368	25.9	19,796	8.4	17,858	7.6
1985	11,163	5.5	10,094	5.0	8,282	28.7	7,556	26.2	19,893	8.3	18,131	7.6
1986	11,690	5.7	10,497	5.1	9,495	32.4	8,790	21.0	21,731	9.0	19,849	8.2
1987	11,128	5.4	9,710	4.7	9,487	31.9	8,272	27.8	21,103	8.7	18,509	7.6
1988	11,066	5.9	9,169	4.4	10,403	34.4	8,895	29.5	22,032	9.0	18,546	7.5

*Homicides/100,000 population.

†Data sources: National Center for Health Statistics (NCHS) Mortality Tapes, Federal Bureau of Investigation (FBI)—Supplementary Homicide Report.

§1988 Data from the FBI do not include Florida or Kentucky.

¶Includes all race categories.

TABLE 2a. Homicides and homicide rates,* by age of victim — United States, 1979–1988†

Year	Age (years) <1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
	Homicide rates — total§										
1979	5.2	2.5	1.0	1.2	14.5	18.2	14.3	10.8	7.1	5.2	10.0
1980	5.9	2.5	0.9	1.4	15.7	19.4	15.0	11.1	7.0	5.5	10.7
1981	6.0	2.6	1.0	1.6	14.7	18.4	14.5	11.3	7.1	5.0	10.3
1982	6.6	2.8	1.0	1.3	13.7	17.2	13.3	10.2	6.5	4.9	9.7
1983	5.2	2.3	0.9	1.2	12.4	15.3	11.9	8.7	6.1	4.5	8.6
1984	6.6	2.4	0.9	1.6	12.0	14.7	11.3	8.5	5.8	4.3	8.4
1985	5.3	2.4	1.0	1.5	12.1	14.7	11.3	8.1	5.7	4.3	8.3
1986	6.5	2.7	0.8	1.5	14.2	16.1	11.4	8.3	5.4	4.5	9.0
1987	7.2	2.3	0.8	1.6	14.0	15.1	10.9	7.8	5.5	4.6	8.7
1988	8.2	2.6	1.0	1.7	15.4	16.0	10.9	7.2	5.2	4.4	9.0
	Homicides — total§										
1979	170	314	165	229	6,156	6,545	3,585	2,488	1,512	1,309	22,550
1980	210	319	153	262	6,647	7,267	3,869	2,530	1,527	1,418	24,278
1981	218	341	156	284	6,172	7,179	3,817	2,548	1,561	1,312	23,646
1982	243	377	166	237	5,695	6,813	3,729	2,285	1,428	1,321	22,358
1983	193	320	144	213	5,037	6,201	3,477	1,948	1,354	1,241	20,191
1984	237	341	155	274	4,819	6,054	3,463	1,916	1,288	1,191	19,796
1985	200	348	167	250	4,772	6,190	3,581	1,828	1,272	1,222	19,893
1986	278	382	134	245	5,522	6,904	3,783	1,895	1,195	1,305	21,731
1987	273	334	141	266	5,354	6,546	3,722	1,803	1,204	1,359	21,103
1988	315	381	179	280	5,771	6,992	3,834	1,727	1,130	1,332	22,032

*Homicides/100,000 population.
†Data source: National Center for Health Statistics Mortality Tapes.
§Includes all race categories.

TABLE 2b. Homicides and homicide rates* for whites, by age of victim — United States, 1979–1988[†]

Year	Age (years) <1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65[†]	Total
	Homicide rates — whites										
1979	3.5	1.8	0.8	1.0	9.5	10.4	8.7	6.9	4.6	4.1	6.4
1980	4.3	1.7	0.7	1.1	10.2	11.5	9.7	7.3	4.8	4.4	7.0
1981	4.6	1.7	0.7	1.1	9.4	10.9	9.3	7.5	4.9	3.8	6.6
1982	4.9	1.8	0.8	1.0	8.8	10.2	8.9	6.8	4.5	3.7	6.3
1983	3.5	1.5	0.7	0.9	7.7	9.4	7.9	6.0	4.2	3.4	5.6
1984	4.5	1.8	0.6	1.2	7.8	9.0	7.6	6.0	4.1	3.1	5.5
1985	4.0	1.8	0.7	1.2	7.4	9.1	7.5	5.7	4.2	3.3	5.5
1986	5.3	1.7	0.5	1.2	8.5	9.6	7.5	5.6	3.9	3.3	5.7
1987	5.1	1.7	0.6	1.0	7.6	9.0	6.9	5.5	4.0	3.5	5.4
1988	5.8	1.9	0.7	1.0	7.8	8.9	6.8	6.0	3.9	3.3	5.3
	Homicides — whites										
1979	96	177	105	145	3,390	3,248	1,905	1,402	886	924	12,334
1980	125	181	100	172	3,626	3,696	2,168	1,468	937	1,030	13,558
1981	137	188	94	170	3,324	3,633	2,134	1,490	957	897	13,066
1982	146	203	103	150	3,057	3,438	2,170	1,331	890	905	12,439
1983	104	166	95	136	2,593	3,237	2,011	1,164	834	846	11,235
1984	133	208	80	170	2,580	3,152	1,993	1,164	814	789	11,127
1985	121	204	95	164	2,427	3,245	2,061	1,109	824	861	11,163
1986	160	194	68	155	2,716	3,444	2,137	1,111	756	876	11,690
1987	156	195	86	134	2,384	3,260	2,026	1,093	774	940	11,128
1988	180	221	108	139	2,373	3,249	2,051	1,048	746	886	11,066

*Homicides/100,000 population.
[†]Data Source: National Center for Health Statistics Mortality Tapes.

Table 2c. Homicides and homicide rates* for blacks, by age of victim — United States, 1979–1988†

Year	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65†	Total
						Age groups					
					Homicide rates — blacks						
1979	14.5	6.9	2.2	3.1	47.0	79.1	61.7	46.2	31.5	17.9	37.6
1980	15.6	6.8	2.0	3.2	50.4	80.1	59.3	44.4	29.6	17.3	38.4
1981	13.2	7.3	2.3	3.9	47.2	75.8	57.2	43.6	29.8	18.3	37.3
1982	15.9	7.7	2.5	2.8	43.8	69.3	50.7	38.9	25.9	17.9	34.3
1983	14.8	6.8	1.8	2.7	40.7	57.9	45.8	31.2	24.5	16.8	30.3
1984	18.7	5.9	2.7	3.6	37.7	55.4	43.4	29.8	22.0	16.6	29.0
1985	13.2	6.4	2.3	2.9	39.7	54.8	42.8	27.9	20.5	14.8	28.7
1986	20.0	8.1	2.2	3.3	47.2	62.4	44.2	29.9	19.7	17.2	32.4
1987	19.1	6.0	1.8	4.6	51.2	58.5	43.5	26.5	19.0	16.4	31.9
1988	21.4	6.9	2.3	5.1	59.0	64.8	44.0	24.5	16.9	16.9	34.4
						Homicides — blacks					
1979	70	132	56	82	2,660	3,168	1,617	1,044	596	368	9,812
1980	84	131	49	85	2,904	3,439	1,625	1,015	569	364	10,283
1981	71	147	57	106	2,730	3,434	1,599	1,003	583	394	10,137
1982	87	159	60	77	2,516	3,264	1,481	901	517	394	9,473
1983	81	142	43	73	2,326	2,821	1,394	729	496	378	8,494
1984	101	125	69	96	2,137	2,780	1,382	705	452	380	8,240
1985	75	136	60	77	2,230	2,827	1,424	669	428	346	8,282
1986	113	174	60	86	2,644	3,300	1,547	730	415	411	9,495
1987	109	130	49	120	2,845	3,162	1,590	659	404	402	9,487
1988	127	152	64	132	3,247	3,567	1,677	627	362	423	10,403

*Homicides/100,000 population.
†Data Source: National Center for Health Statistics Mortality Tapes.

TABLE 2d. Homicides and homicide rates* for white males, by age of victim — United States, 1979–1988†

Year	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
	Homicide rates — white males										
1979	4.2	1.8	0.8	1.1	14.4	16.8	13.9	11.2	7.4	5.9	9.9
1980	4.3	2.0	0.7	1.1	15.5	18.7	15.5	11.9	7.8	6.7	10.9
1981	4.5	1.6	0.7	1.2	14.4	17.5	15.1	12.1	7.9	5.3	10.4
1982	5.6	1.9	0.8	0.9	13.1	16.1	13.9	10.8	7.1	5.2	9.6
1983	3.2	1.7	0.8	1.1	11.5	14.8	12.5	9.1	6.4	4.7	8.6
1984	5.0	1.9	0.5	1.2	11.1	14.1	11.8	9.4	6.3	4.3	8.3
1985	3.7	1.9	0.7	1.4	11.2	13.9	11.5	8.6	6.3	4.5	8.2
1986	5.4	1.9	0.5	1.2	12.5	14.6	11.6	8.6	6.0	4.4	8.6
1987	6.0	1.8	0.6	1.0	11.2	13.2	10.2	8.3	6.3	4.5	7.9
1988	5.6	2.2	0.8	1.3	11.5	13.2	10.4	7.6	6.0	4.2	7.9
	Homicides — white males										
1979	58	93	58	89	2,622	2,622	1,504	1,103	669	536	9,392
1980	64	106	50	88	2,800	3,015	1,712	1,161	718	628	10,381
1981	69	90	50	82	2,575	2,923	1,713	1,167	729	510	9,941
1982	86	108	51	68	2,303	2,724	1,679	1,039	659	511	9,260
1983	50	97	52	83	1,979	2,548	1,568	872	600	469	8,355
1984	75	115	35	90	1,874	2,479	1,547	900	588	439	8,171
1985	58	111	49	101	1,850	2,478	1,563	822	588	462	8,122
1986	84	115	38	84	2,034	2,664	1,640	833	555	466	8,567
1987	94	107	47	71	1,786	2,429	1,502	820	579	491	7,979
1988	89	131	56	86	1,784	2,446	1,561	782	544	466	7,994

*Homicides/100,000 population.
†Data Source: National Center for Health Statistics Mortality Tapes.

TABLE 2e. Homicides and homicide rates* for black males, by age of victim — United States, 1979–1988†

Year	Age (years) <1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
	Homicide rates — black males										
1979	18.0	6.2	2.3	4.1	76.9	143.1	113.9	85.5	57.6	30.0	64.5
1980	18.5	7.2	1.9	3.9	83.9	143.0	109.5	83.6	55.3	31.0	66.2
1981	12.2	8.9	2.8	5.2	78.8	136.2	106.5	82.7	52.6	34.7	64.6
1982	17.4	8.9	2.9	3.7	73.3	124.5	92.3	72.8	48.0	31.6	59.1
1983	14.1	7.2	2.1	4.0	66.7	101.3	83.1	57.8	46.6	29.2	51.4
1984	20.6	5.0	2.4	4.0	61.5	96.3	78.2	57.1	40.6	29.6	48.7
1985	16.0	6.5	2.3	4.1	66.1	94.4	76.4	51.1	37.8	25.2	48.4
1986	22.8	9.3	1.9	4.6	79.1	108.1	79.5	56.3	35.4	29.2	55.0
1987	19.4	4.8	2.0	6.8	85.5	99.0	78.5	46.0	32.8	28.5	53.3
1988	19.3	7.5	2.7	5.7	101.8	108.9	79.3	45.3	29.1	27.8	58.1
	Homicides — black males										
1979	43	60	29	55	2,129	2,653	1,346	868	488	252	7,938
1980	50	70	24	53	2,365	2,854	1,362	858	475	264	8,385
1981	33	90	35	71	2,231	2,877	1,349	853	461	302	8,312
1982	48	92	36	51	2,066	2,739	1,223	756	429	280	7,730
1983	39	77	26	54	1,870	2,307	1,146	607	425	264	6,822
1984	56	54	31	54	1,710	2,264	1,131	607	376	273	6,563
1985	46	70	30	55	1,825	2,285	1,156	550	355	237	6,616
1986	65	102	26	60	2,184	2,688	1,265	615	337	280	7,634
1987	56	53	28	89	2,346	2,522	1,303	514	315	280	7,518
1988	58	84	38	76	2,762	2,827	1,375	518	282	278	8,314

*Homicides/100,000 population.
†Data Source: National Center for Health Statistics Mortality Tapes.

TABLE 2f. Homicides and homicide rates* for white females, by age of victim — United States, 1979–1988†

	Age (years)										
Year	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
	Homicide rates — white females										
1979	2.9	1.7	0.7	0.8	4.3	4.1	3.6	2.9	2.1	2.9	3.0
1980	4.3	1.5	0.8	1.1	4.7	4.3	4.0	3.0	2.1	2.9	3.2
1981	4.7	1.9	0.7	1.2	4.3	4.3	3.7	3.2	2.2	2.7	3.1
1982	4.1	1.8	0.8	1.1	4.4	4.3	4.0	2.9	2.2	2.7	3.1
1983	3.7	1.3	0.7	0.7	3.7	4.1	3.5	2.9	2.2	2.6	2.8
1984	4.1	1.7	0.7	1.2	4.3	3.9	3.4	2.7	2.2	2.3	2.9
1985	4.3	1.7	0.7	0.9	3.6	4.4	3.6	2.9	2.3	2.6	2.9
1986	5.2	1.4	0.4	1.1	4.3	4.4	3.5	2.8	1.9	2.6	3.0
1987	4.2	1.5	0.6	1.0	3.9	4.6	3.5	2.7	1.9	2.8	3.0
1988	6.0	1.6	0.7	0.8	3.9	4.4	3.2	2.5	2.0	2.6	2.9
	Homicides — white females										
1979	38	84	47	56	768	626	401	299	217	388	2,942
1980	61	75	50	84	826	681	456	307	219	402	3,177
1981	68	98	44	88	749	710	421	323	228	387	3,125
1982	60	95	52	82	754	714	491	292	231	394	3,179
1983	54	69	43	53	614	689	443	292	234	377	2,880
1984	58	93	45	80	706	673	446	264	226	350	2,956
1985	63	93	46	63	577	767	498	287	236	399	3,041
1986	76	79	30	71	682	780	497	278	201	410	3,123
1987	62	88	39	63	598	831	524	273	195	449	3,149
1988	91	90	52	53	589	803	490	266	202	420	3,072

*Homicides/100,000 population.
†Data Source: National Center for Health Statistics Mortality Tapes.

TABLE 2g. Homicides and homicide rates* for black females, by age of victim — United States, 1979–1988†

Year	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
	Homicide rates — black females										
1979	11.3	7.7	2.2	2.0	18.3	23.9	18.8	14.1	10.3	9.5	13.6
1980	12.6	6.4	2.0	2.4	18.3	25.4	17.6	12.5	8.8	8.0	13.4
1981	14.2	5.7	1.8	2.6	16.9	23.0	16.4	11.8	11.3	7.2	12.7
1982	14.3	6.6	2.0	1.9	15.4	20.9	16.2	11.4	8.0	8.7	12.0
1983	15.5	6.3	1.4	1.4	15.7	19.8	14.9	9.5	6.4	8.5	11.3
1984	16.9	6.8	3.1	3.2	14.8	19.3	14.4	7.5	6.7	7.8	11.2
1985	10.3	6.3	2.3	1.7	14.2	19.8	14.7	9.0	6.4	7.8	11.0
1986	17.2	6.8	2.6	2.0	16.2	21.9	14.8	8.5	6.7	9.1	12.1
1987	18.7	7.2	1.6	2.4	17.7	22.4	14.4	10.6	7.6	8.3	12.6
1988	23.6	6.3	1.9	4.4	17.4	25.5	14.6	7.7	6.8	9.7	13.2
	Homicides — black females										
1979	27	72	27	27	531	515	271	176	108	116	1,874
1980	34	61	25	32	539	585	263	157	94	100	1,898
1981	38	57	22	35	499	557	250	150	122	92	1,825
1982	39	67	24	26	450	525	258	145	88	114	1,743
1983	42	65	17	19	456	514	248	122	71	114	1,672
1984	45	71	38	42	427	516	251	98	76	107	1,677
1985	29	66	30	22	405	542	268	119	73	109	1,666
1986	48	72	34	26	460	612	282	115	78	131	1,861
1987	53	77	21	31	499	640	287	145	89	122	1,969
1988	69	68	26	56	485	740	302	109	80	145	2,089

*Homicides/100,000 population.
†Data Source: National Center for Health Statistics Mortality Tapes.

TABLE 3. Ten leading causes of death, by years of potential life lost (YPLL) for persons <65 years of age, by race — United States, 1988*

	White			Black			Total[†]		
Rank	**Cause**	**YPLL**	**%**	**Cause**	**YPLL**	**%**	**Cause**	**YPLL**	**%**
1	Unintentional injuries	1,921,259	21	Perinatal period	429,545	15	Unintentional injuries	2,363,827	19
2	Malignant neoplasms	1,476,618	16	Unintentional injuries	368,120	13	Malignant neoplasms	1,795,163	15
3	Heart disease	1,106,364	12	**Homicide**	358,336	12	Heart disease	1,447,150	12
4	Perinatal period	726,180	8	Heart disease	316,257	11	Perinatal period	1,182,628	10
5	Suicide	603,678	7	Malignant neoplasms	280,037	10	**Homicide**	712,1226	6
6	Congenital anomalies	532,958	6	HIV[§]	150,845	5	Suicide	681,196	6
7	**Homicide**	335,095	4	Congenital anomalies	114,547	4	Congenital anomalies	670,867	5
8	HIV	298,251	3	Cerebro-vascular disease	73,490	3	HIV	452,694	4
9	Liver disease	175,008	2	Suicide	59,736	2	Cerebro-vascular disease	244,980	2
10	Cerebro-vascular disease	164,567	2	Pneumonia-influenza	58,701	2	Liver disease	238,399	2
All other causes		1,723,461	19	All other causes	718,763	25	All other causes	2,513,437	20

*Data Source: National Center for Health Statistics Mortality Tapes.
[†]Includes all race categories.
[§]HIV = Human immunodeficiency virus.

TABLE 4a. Ten leading causes of death, by age group — United States, 1988*

	Age (years)†										
Rank	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
1	Perinatal period 18,036	Unintentional injuries 2,858	Unintentional injuries 2,102	Unintentional injuries 2,113	Unintentional injuries 18,507	Unintentional injuries 16,728	Malignant neoplasms 15,581	Malignant neoplasms 38,766	Malignant neoplasms 97,656	Heart disease 627,494	Heart disease 765,048
2	Congenital anomalies 8,141	Congenital anomalies 913	Malignant neoplasms 586	Malignant neoplasms 510	**Homicide** **5,771**	**Homicide** **6,992**	Heart disease 12,070	Heart disease 31,758	Heart disease 87,514	Malignant neoplasms 324,187	Malignant neoplasms 485,022
3	Unintentional injuries 936	Malignant neoplasms 542	Congenital anomalies 281	**Homicide** **280**	Suicide 4,929	Suicide 6,710	Unintentional injuries 11,551	Unintentional injuries 7,514	Cerebrovascular disease 11,196	Cerebrovascular disease 130,745	Cerebrovascular disease 150,508
4	Heart disease 871	**Homicide** **381**	**Homicide** **179**	Suicide 237	Malignant neoplasms 1,894	HIV 6,036	HIV 6,184	Liver disease 4,780	Bronchitis, emphysema, asthma 10,659	Bronchitis, emphysema, asthma 68,614	Unintentional injuries 96,968
5	Pneumonia & influenza 641	Heart disease 352	Heart disease 146	Congenital anomalies 218	Heart disease 1,090	Malignant neoplasms 5,211	Suicide 5,205	Cerebrovascular disease 4,630	Unintentional injuries 7,663	Pneumonia & influenza 68,343	Bronchitis, emphysema asthma 82,842
6	**Homicide** **315**	Pneumonia & influenza 186	Pneumonia & influenza 66	Heart disease 178	HIV 535	Heart disease 3,575	**Homicide** **3,834**	Suicide 3,532	Liver disease 6,980	Diabetes 29,547	Pneumonia & influenza 77,651
7	Septicemia 245	(Tied) Meningitis 129 Perinatal period 129	Benign neoplasm 54	Bronchitis, emphysema, asthma 71	Congenital anomalies 474	Liver disease 1,057	Liver disease 3,563	Diabetes 2,502	Diabetes 6,109	Unintentional injuries 26,966	Diabetes 40,368

TABLE 4a. Ten leading causes of death, by age group — United States, 1988* — Continued

	Age (years)†										
Rank	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
8	Nephritis 218		HIV 37	Pneumonia 61	(Tied) Cerebro-vascular disease 266 Pneumonia & influenza 266	Cerebro-vascular disease 969	Cerebro-vascular disease 2,423	HIV 2,352	Pneumonia & influenza 4,124	Athero-sclerosis 21,055	Suicide 30,388
9	Meningitis 205	HIV 114	(Tied) Cerebro-vascular disease 32 Bronchitis, emphysema, asthma 32	Benign neoplasms 49		Pneumonia & influenza 266	Diabetes 1,395	Bronchitis, emphysema, asthma 2,259	Suicide 3,406	Nephritis 18,453	Liver disease 26,401
10	Cerebro-vascular disease 152	Septicemia 89		Cerebro-vascular disease 40	Bronchitis 178	Diabetes 656	Pneumonia & influenza 1,348	Pneumonia & influenza 1,771	Nephritis 2,058	Septicemia 16,876	Nephritis 22,391

*Data Source: National Center for Health Statistics Mortality Tapes.
†Cause and number of deaths are represented in each cell.
§HIV = Human immunodeficiency virus.

TABLE 4b. Ten leading causes of death* for white males, by age group — United States, 1988*

	Age (years)										
Rank	**<1**	**1–4**	**5–9**	**10–14**	**15–24**	**25–34**	**35–44**	**45–54**	**55–64**	**≥65**	**Total**
1	Perinatal period 6,407	Unintentional injuries 1,259	Unintentional injuries 984	Unintentional injuries 1,140	Unintentional injuries 12,147	Unintentional injuries 10,804	Unintentional injuries 7,083	Heart disease 19,082	Heart disease 51,354	Heart disease 261,302	Heart disease 341,519
2	Congenital anomalies 3,474	Congenital anomalies 417	Malignant neoplasms 289	Malignant neoplasms 244	Suicide 3,618	Suicide 4,746	Heart disease 6,962	Malignant neoplasms 15,714	Malignant neoplasms 46,221	Malignant neoplasms 153,159	Malignant neoplasms 224,514
3	Unintentional injuries 360	Malignant neoplasms 236	Congenital anomalies 117	Suicide 146	**Homicide** **1,784**	HIV[§] 3,599	Malignant neoplasms 5,556	Unintentional injuries 4,396	Bronchitis, emphysema, asthma 5,473	Cerebrovascular disease 43,110	Unintentional injuries 54,435
4	Heart disease 339	**Homicide** **131**	(Tied) **Homicide** **56** Heart disease 56	Congenital anomalies 108	Malignant neoplasms 916	**Homicide** **2,446**	HIV 4,015	Liver disease 2,669	Cerebrovascular disease 4,577	Bronchitis, emphysema, asthma 37,971	Cerebrovascular disease 50,692
5	Pneumonia & influenza 239	Heart disease 117		(Tied) **Homicide** **86** Heart disease 86	Heart disease 479	Malignant neoplasms 2,130	Suicide 3,629	Suicide 2,374	Unintentional injuries 4,274	Pneumonia & influenza 28,174	Bronchitis, emphysema, asthma 44,827
6	Septicemia 91	Pneumonia & influenza 66	Pneumonia & influenza 30		HIV 282	Heart disease 1,698	Liver disease 1,890	HIV 1,682	Liver disease 4,044	Unintentional injuries 11,894	Pneumonia & influenza 32,262

TABLE 4b. Ten leading causes of death* for white males, by age group — United States, 1988* — Continued

	Age (years)										
Rank	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
7	**Homicide**	Meningitis	Benign neoplasms	Pneumonia & influenza	Congenital anomalies	Liver disease	**Homicide**	Cerebro-vascular disease	Suicide	Diabetes	Suicide
	89	49	22	27	210	494	**1,561**	1,642	2,450	9,724	21,980
8	Nephritis	Perinatal period	Bronchitis, emphysema, asthma	Bronchitis, emphysema, asthma	Cerebro-vascular disease	Cerebro-vascular disease	Cerebro-vascular disease	Diabetes	Diabetes	Nephritis	Liver disease
	82	48	13	21	116	331	822	1,002	2,293	7,322	14,381
9	Meningitis	Septicemia	Perinatal period	Benign neoplasms	Pneumonia & influenza	Pneumonia & influenza	Diabetes	Bronchitis, emphysema, asthma	Pneumonia & influenza	Arthero-sclerosis	Diabetes
	74	32	12	19	99	318	622	962	2,000	6,969	14,008
10	Cerebro-vascular disease	Benign neoplasms	Septicemia	Cerebro-vascular disease	Diabetes	Diabetes	Pneumonia & influenza	**Homicide**	Nephritis	Septicemia	HIV
	50	29	11	17	50	303	557	**782**	801	5,751	10,479

*Data source: National Center for Health Statistics Mortality Tapes.
†Cause and number of deaths are represented in each cell.
§HIV = Human immunodeficiency virus.

TABLE 4c. Ten leading causes of death* for black males, by age group — United States, 1988*

	Age (years)										
Rank	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
1	Perinatal period 3,660	Unintentional injuries 400	Unintentional injuries 315	Unintentional injuries 263	**Homicide 2,762**	**Homicide 2,827**	Heart disease 1,961	Heart disease 4,041	Heart disease 8,072	Heart disease 24,331	Heart disease 39,584
2	Congenital anomalies 747	**Homicide 84**	Malignant neoplasms 46	**Homicide 76**	Unintentional injuries 1,592	Unintentional injuries 1,920	HIV[§] 1,592	Malignant neoplasms 3,460	Malignant neoplasms 7,266	Malignant neoplasms 17,749	Malignant neoplasms 30,321
3	Unintentional injuries 160	Congenital anomalies 79	**Homicide 38**	Malignant neoplasms 38	Suicide 394	HIV 1,632	Unintentional injuries 1,551	Unintentional injuries 991	Cerebrovascular disease 1,419	Cerebrovascular disease 5,228	Unintentional injuries 9,608
4	Heart disease 129	Heart disease 51	Congenital anomalies 35	Heart disease 26	Heart disease 214	Heart disease 716	**Homicide 1,375**	Cerebrovascular disease 763	Unintentional injuries 867	Bronchitis, emphysema, asthma 2,520	**Homicide 8,314**
5	Pneumonia & influenza 123	Malignant neoplasms 38	Heart disease 22	(Tied) Congenital anomalies 17 Suicide 17	Malignant neoplasms 169	Suicide 574	Malignant neoplasms 1,180	Liver disease 652	Bronchitis emphysema, asthma 631	Pneumonia & influenza 2,375	Cerebrovascular disease 8,098
6	**Homicide 58**	HIV 36	Anemias 10		HIV 161	Malignant neoplasms 363	Liver disease 640	**Homicide 518**	Diabetes 585	Unintentional injuries 1,523	HIV 4,202

TABLE 4c. Ten leading causes of death* for black males, by age group — United States, 1988* — Continued

	Age (years)										
Rank	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
7	Septicemia 37	Perinatal Period 29	(Tied) HIV 9 Bronchitis, emphysema, asthma 9	Bronchitis emphysema, asthma 15	Congenital anomalies 49	Pneumonia & influenza 201	Cerebro-vascular disease 449	HIV 517	Liver disease 576	Diabetes 1,430	Pneumonia & influenza 4,047
8	Meningitis 34	Meningitis 27		Pneumonia & influenza 8	Bronchitis emphysema, asthma 44	Liver disease 193	Pneumonia & influenza 327	Pneumonia & influenza 401	Pneumonia & influenza 538	Nephritis 1,190	Perinatal period 3,693
9	Nephritis 29	Pneumonia & influenza 23	Pneumonia & influenza 8	Anemias 4	Pneumonia & influenza 42	Cerebro-vascular disease 175	Suicide 285	Diabetes 315	Nephritis 297	Septicemia 1,050	Bronchitis, emphysema, asthma 3,644
10	Cerebro-vascular disease 28	Anemias 17	Benign neoplasms 4	Cerebro-vascular disease 3	Anemias 34	Diabetes 83	Diabetes 210	Bronchitis emphysema, asthma 225	**Homicide** **282**	Athero-sclerosis 639	Diabetes 2,640

*Data Source: National Center for Health Statistics Mortality Tapes.
†Cause and number of deaths are represented in each cell.
§HIV = Human immunodeficiency virus.

TABLE 4d. Ten leading causes of death* for white females, by age group — United States, 1988*

Rank	Age (years) <1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
1	Perinatal period 4,653	Unintentional injuries 819	Unintentional injuries 544	Unintentional injuries 508	Unintentional injuries 3,742	Unintentional injuries 2,903	Malignant neoplasms 6,996	Malignant neoplasms 16,033	Malignant neoplasms 37,493	Heart disease 306,409	Heart disease 337,007
2	Congenital anomalies 2,968	Congenital anomalies 310	Malignant neoplasms 186	Malignant neoplasms 171	Suicide 690	Malignant neoplasms 2,080	Unintentional injuries 2,112	Heart disease 5,777	Heart disease 21,464	Malignant neoplasms 136,791	Malignant neoplasms 200,626
3	Unintentional injuries 278	Malignant neoplasms 210	Congenital anomalies 96	Congenital anomalies 72	Malignant neoplasms 627	Suicide 1,098	Heart disease 1,892	Unintentional injuries 1,650	Bronchitis, emphysema, asthma 4,061	Cerebrovascular disease 73,022	Cerebrovascular disease 79,383
4	Heart disease 255	Heart disease 125	(Tied) **Homicide** **52** Heart disease 52	**Homicide** **53**	**Homicide** **589**	**Homicide** **803**	Suicide 1,121	Cerebrovascular disease 1,472	Cerebrovascular disease 3,723	Pneumonia & influenza 34,818	Pneumonia & influenza 37,846
5	Pneumonia & influenza 148	**Homicide** **90**		Suicide 49	Heart disease 255	Heart disease 705	Cerebrovascular disease 696	Liver disease 992	Diabetes 2,279	Bronchitis, emphysema, asthma 26,541	Bronchitis, emphysema, asthma 31,308
6	**Homicide** **91**	Pneumonia & influenza 68	Benign neoplasm 23	Heart disease 46	Congenital anomalies 183	HIV[§] 308	Liver disease 612	Suicide 911	Unintentional injuries 2,028	Diabetes 14,981	Unintentional injuries 26,656
7	Nephritis 66	Meningitis 40	(Tied) Cerebrovascular disease 19	(Tied) Benign neoplasms 19	Cerebrovascular disease 91	Cerebrovascular disease 282	**Homicide** **490**	Bronchitis, emphysema, asthma 861	Liver disease 1,902	Atherosclerosis 12,447	Diabetes 18,684

TABLE 4d. Ten leading causes of death* for white females, by age group — United States, 1988* — Continued

	Age (years)										
Rank	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
			Pneumonia & influenza 19	Pneumonia & influenza 19							
8	Septicemia 64	Perinatal period 38			Pneumonia & influenza 84	Liver disease 207	Diabetes 395	Diabetes 777	Pneumonia & influenza 1,233	Unintentional injuries 12,064	Athero-sclerosis 12,732
9	Meningitis 61	Septicemia 29	Septicemia 13	Bronchitis, emphysema, asthma 16	Complicated pregnancy 56	Diabetes 198	Pneumonia & influenza 289	Pneumonia & influenza 434	Suicide 796	Septicemia 8,448	Septicemia 9,673
10	Cerebro-vascular disease 43	Benign neoplasms 24	Meningitis 10	Cerebro-vascular disease 12	Bronchitis, emphysema, asthma 51	Pneumonia & influenza 191	HIV 206	Homicide 266	Septicemia 644	Nephritis 8,068	Nephritis 9,129

*Data Source: National Center for Health Statistics Mortality Tapes.
†Cause and number of deaths are represented in each cell.
§HIV = Human immunodeficiency virus.

TABLE 4e. Ten leading causes of death* for black females, by age group — United States, 1988*

	Age (years)										
Rank	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
1	Perinatal period 2,906	Unintentional injuries 266	Unintentional injuries 182	Unintentional injuries 124	**Homicide** **485**	**Homicide** **740**	Malignant neoplasms 1,476	Malignant neoplasms 2,771	Heart disease 5,515	Heart disease 30,236	Heart disease 39,882
2	Congenital anomalies 663	Congenital anomalies 69	Malignant neoplasms 37	**Homicide** **56**	Unintentional injuries 417	Unintentional injuries 610	Heart disease 1,053	Heart disease 2,369	Malignant neoplasms 5,313	Malignant neoplasms 13,312	Malignant neoplasms 23,647
3	Unintentional injuries 121	**Homicide** **68**	**Homicide** **26**	Malignant neoplasms 38	Malignant neoplasms 136	Malignant neoplasms 509	Unintentional injuries 470	Cerebrovascular disease 607	Cerebrovascular disease 1,237	Cerebrovascular disease 7,924	Cerebrovascular disease 10,381
4	Heart disease 117	Heart disease 45	Congenital anomalies 19	Congenital anomalies 17	Heart disease 122	HIV[§] 448	Cerebrovascular disease 384	Diabetes 336	Diabetes 813	Diabetes 2,948	Diabetes 4,332
5	Pneumonia & influenza 101	Malignant neoplasms 41	(Tied) HIV 12 Heart disease 12	Bronchitis, emphysema, asthma 16	Suicide 71	Heart disease 383	HIV 320	Liver disease 313	Bronchitis, emphysema, asthma 397	Pneumonia & influenza 2,270	Unintentional injuries 3,879
6	**Homicide** **69**	HIV 30		Heart disease 14	Complicated Pregnancy 50	Cerebrovascular disease 155	Liver disease 317	Unintentional injuries 292	Liver disease 320	Nephritis 1,643	Pneumonia & influenza 3,144
7	Septicemia 45	Pneumonia & influenza 22	Pneumonia & influenza 6	Suicide 12	HIV 48	Pneumonia & influenza 131	**Homicide** **302**	Bronchitis, emphysema, asthma 181	Nephritis 307	Septicemia 1,454	Perinatal period 2,921

TABLE 4e. Ten leading causes of death* for black females, by age group — United States, 1988* — Continued

	Age (years)										
Rank	<1	1–4	5–9	10–14	15–24	25–34	35–44	45–54	55–64	≥65	Total
8	Nephritis 35	Anemias 17	Anemias 5	Meningitis 9	(Tied) Anemias 33 Pneumonia & influenza 33	Liver disease 114	Pneumonia & influenza 154	Pneumonia & influenza 147	Unintentional injuries 304	Unintentional injuries 1,090	Nephritis 2,249
9	Meningitis 28	Meningitis 13	(Tied) Septicemia 4 Benign neoplasms 4 Bronchitis, emphysema, asthma 4 Meningococcal 4	Benign neoplasms 8 8		Suicide 11	Diabetes 149	Nephritis 134	Pneumonia & influenza 276	Bronchitis, emphysema, asthma 1,046	**Homicide** **2,089**
10	Cerebrovascular 24	(Tied) Septicemia 10 Bronchitis, emphysema, asthma 10 Perinatal period 10		(Tied) Anemias 7 Cerebrovascular disease 7	Cerebrovascular disease 30	Diabetes 68	Bronchitis, emphysema, asthma 96	**Homicide** **109**	Septicemia 232	Atherosclerosis 880	Septicemia 2,011

*Data Source: National Center for Health Statistics Mortality Tapes.
†Cause and number of deaths are represented in each cell.
§HIV = Human immunodeficiency virus.

TABLE 5. Number, rate,* and relative rank of homicides, by state — United States, 1986–1988†

State of occurrence	Number of deaths	Crude rate per 100,000	Crude rank	Adjusted rate§ per 100,000	Adjusted rank
1. Alabama	1,382	11.3	9	8.4	16
2. Alaska	120	7.5	22	6.6	26
3. Arkansas	685	9.6	15	8.9	14
4. Arizona	974	9.6	16	11.1	6
5. California	9,165	11.1	11	11.8	4
6. Colorado	633	6.4	23	7.6	20
7. Connecticut	484	5.0	32	5.8	34
8. Delaware	102	5.3	20	4.9	40
9. Florida	4,714	13.1	3	12.6	2
10. Georgia	2,472	13.2	2	9.2	12
11. Hawaii	132	4.1	37	4.9	39
12. Idaho	112	3.7	39	10.0	9
13. Illinois	3,310	9.5	17	8.2	18
14. Indiana	918	5.5	28	6.5	28
15. Iowa	169	2.0	49	3.5	44
16. Kansas	319	4.3	35	5.4	35
17. Kentucky	708	6.3	24	6.8	25
18. Louisiana	1,795	13.4	1	8.8	15
19. Maine	86	2.4	47	2.2	48
20. Maryland	1,313	9.7	14	6.5	27
21. Massachusetts	658	3.8	38	5.3	37
22. Michigan	3,309	12.0	6	10.9	7
23. Minnesota	356	2.8	45	5.9	33
24. Mississippi	939	11.9	7	7.9	19
25. Missouri	1,436	9.4	18	9.7	10
26. Montana	103	4.2	36	4.2	43
27. Nebraska	167	3.5	43	5.9	32
32. Nevada	333	11.1	10	11.4	5
28. New Hampshire	75	2.4	48	2.0	49
29. New Jersey	1,132	4.9	33	4.6	41
31. New Mexico	554	12.3	5	12.9	1
30. New York	6,159	11.5	8	10.5	8
33. North Carolina	1,764	9.2	19	7.3	23
34. North Dakota	36	1.8	50	1.4	50
35. Ohio	1,647	5.1	31	5.5	38
36. Oklahoma	850	8.6	20	9.1	13
37. Oregon	488	6.0	25	8.3	17
38. Pennsylvania	1,980	5.5	27	6.1	29
39. Rhode Island	136	4.6	34	6.0	31
40. South Carolina	1,012	9.9	13	7.5	21
41. South Dakota	79	3.7	40	2.4	46
42. Tennessee	1,568	10.8	12	9.5	11
43. Texas	6,416	12.7	4	12.2	3
44. Utah	177	3.5	42	4.4	42
45. Vermont	43	2.6	46	2.2	47
46. Virginia	1,403	7.9	21	6.1	30
47. Washington	746	5.5	29	7.1	24
48. West Virginia	340	6.0	26	7.5	22
49. Wisconsin	507	3.5	41	5.6	35
50. Wyoming	51	3.4	44	3.3	45

*Homicides/100,000 population.
†Data source: National Center for Health Statistics Mortality Tapes.
§Adjusted for age, race, and sex.

TABLE 6. Percentage of homicide victims, by type of weapon used — United States, 1988*

Weapon used	Total	Total white	Total black	Total male	White male	Black male	Total female	White female	Black female
Total firearms	**61.6**	**58.3**	**65.4**	**67.0**	**63.4**	**70.6**	**45.3**	**46.9**	**44.7**
Handgun	46.2	41.5	51.5	50.7	45.4	55.7	32.7	31.7	34.6
Rifle	4.2	5.5	2.8	4.3	5.7	2.9	4.0	5.1	2.1
Shotgun	6.3	7.2	5.4	6.6	7.5	5.8	5.5	6.6	4.1
Other firearm	4.9	4.1	5.7	5.4	4.8	6.2	3.1	2.5	0.1
Knife/cutting instrument	18.8	18.4	19.2	18.2	18.1	18.3	20.4	19.0	22.8
Undetermined	3.3	4.0	2.4	2.5	3.2	1.9	5.4	6.0	4.3
Other weapon†	16.3	19.3	13.0	12.3	15.4	9.2	28.8	29.1	28.2
Total§	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**

*Data source: Federal Bureau of Investigation (FBI)—Supplementary Homicide Report.
†Including bodily force.
§1988 FBI data do not include Florida or Kentucky.

TABLE 7. Percentage of homicide victims and relationship of victim to assailant, by race — United States, 1988*†

Relationship	Total	Total white	Total black	Total male	White male	Black male	Total female	White female	Black female
Family	14.6	17.4	11.6	9.7	10.8	8.7	29.6	33.9	22.9
Acquaintance	39.4	37.6	41.7	40.9	40.0	42.1	35.0	31.5	40.4
Stranger	14.4	17.4	11.1	16.3	20.1	12.5	8.6	10.5	5.7
Undetermined	31.6	27.7	35.7	33.1	29.1	36.8	26.8	24.1	31.1
Total	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**

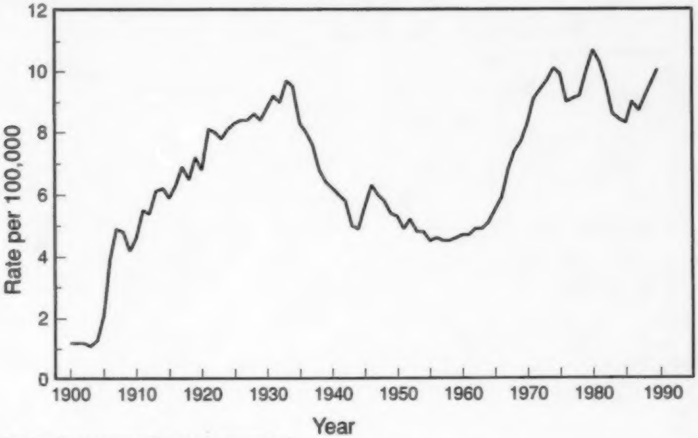
*Data source: Federal Bureau of Investigation (FBI)—Supplementary Homicide Report.
†1988 FBI data do not include Florida or Kentucky.

TABLE 8. Percentage of homicide victims and circumstance, by race — United States, 1988*†

Circumstance	Total	Total white	Total black	Total male	White male	Black male	Total female	White female	Black female
Conflicts§	35.8	34.8	37.0	36.7	36.5	37.0	33.2	30.7	36.9
Drugs	6.5	4.1	9.1	7.7	5.2	10.3	2.7	1.5	4.6
Gangs	2.0	1.7	2.3	2.5	2.2	2.7	0.5	0.3	0.6
Felony	14.0	17.3	10.3	12.9	16.2	9.6	17.3	19.8	13.3
Other	13.1	15.4	10.6	10.6	11.9	9.2	21.0	24.3	16.4
Reverse felony¶	3.1	3.5	2.7	4.0	4.8	3.3	0.2	0.2	0.3
Unknown	25.5	23.2	27.9	25.65	23.2	27.9	25.0	23.3	27.9
Total	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**	**100.0**

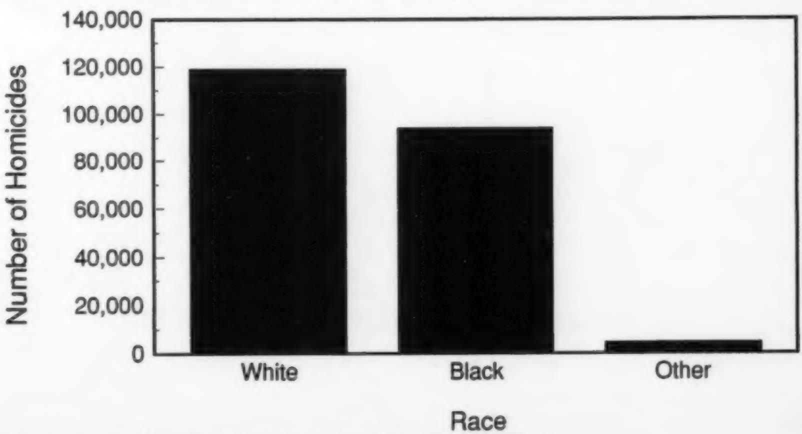
*Data source: Federal Bureau of Investigation—Supplementary Homicide Report.
†1988 FBI data do not include Florida or Kentucky.
§Includes the following categories: lovers' triangle, children killed by babysitter, brawl under the influence of alcohol, argument over money, and other arguments.
¶Includes the following categories: felon killed by police, felon killed by citizen.

FIGURE 1. Homicide rate — United States, 1900*–1990



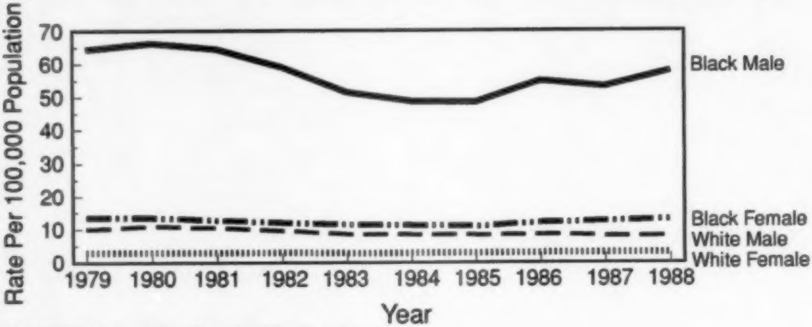
*1933 is the first year all states reported.
Data source: National Center for Health Statistics Mortality Tapes.

FIGURE 2. Number of homicides, by race of victim — United States, 1979–1988



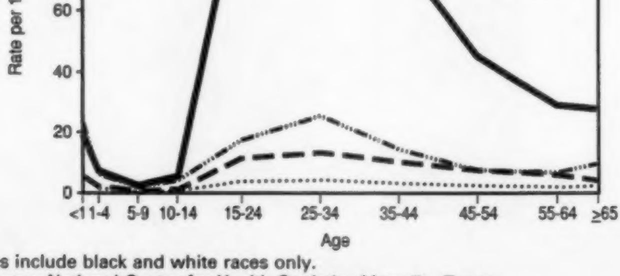
Data source: National Center for Health Statistics Mortality Tapes.

FIGURE 3. Homicide rate, by race* and sex of victim — United States, 1979–1988



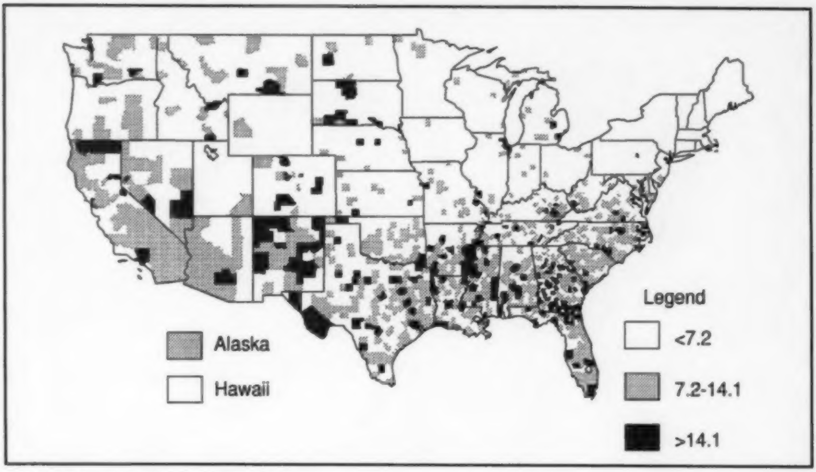
*Analyses include black and white races only.
Data source: National Center for Health Statistics Mortality Tapes.

FIGURE 4. Homicide rates, by age group, race,* and sex — United States, 1988



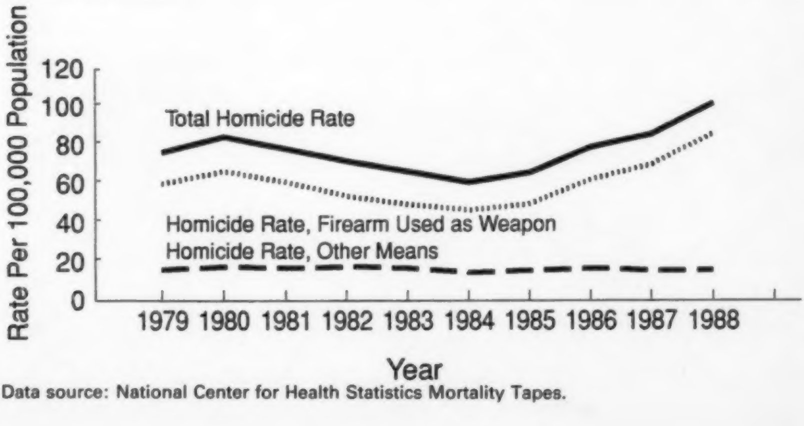
*Analyses include black and white races only.
Data source: National Center for Health Statistics Mortality Tapes.

FIGURE 5. Homicide rates,* by county — United States, 1986–1988



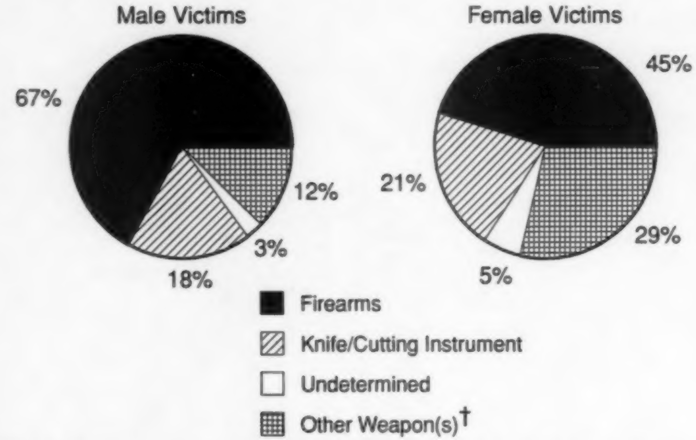
*Homicides per 100,000 population.

FIGURE 6. Homicide rates for black males ages 15–24, by use of firearms — United States, 1979–1988



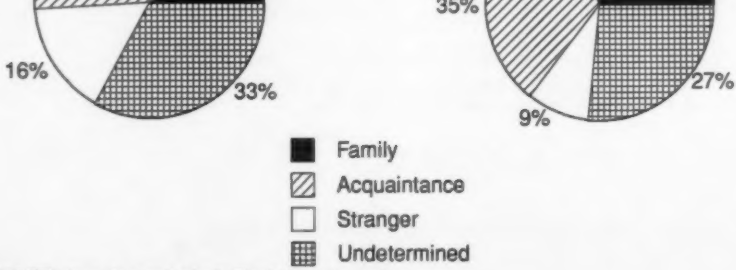
Data source: National Center for Health Statistics Mortality Tapes.

FIGURE 7. Percentage of homicides, by weapon used and sex of victim — United States, 1988*



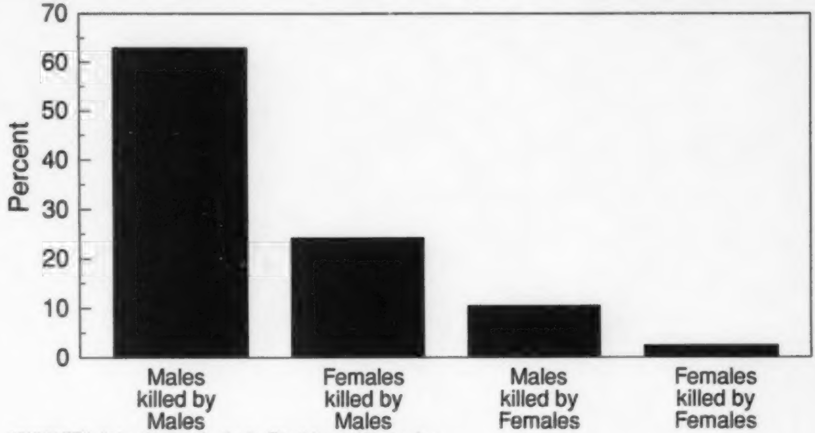
*1988 Federal Bureau of Investigation (FBI) data do not include Florida or Kentucky.
†Including bodily force.
Data source: FBI—Supplementary Homicide Report.

FIGURE 8. Percentage of homicides, by relationship of victim to assailant and sex of victim — United States, 1988*



*1988 FBI data do not include Florida or Kentucky.
Data source: Federal Bureau of Investigation (FBI)—Supplementary Homicide Report.

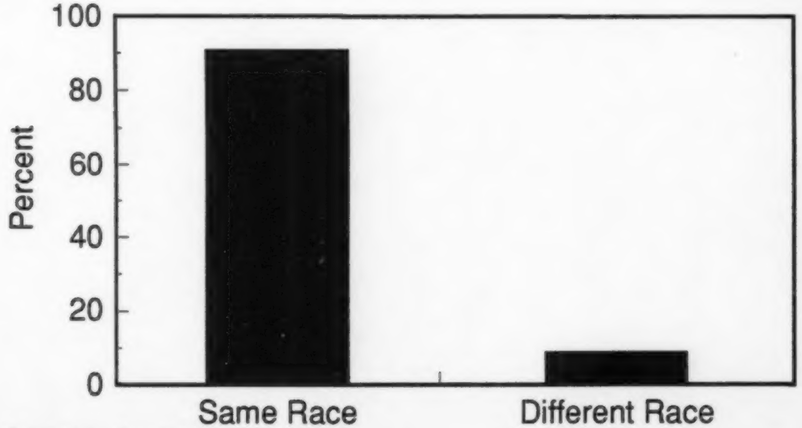
FIGURE 9. Homicides, by sex of victim and offender — United States, 1988*



*1988 FBI data do not include Florida or Kentucky.

Data source: Federal Bureau of Investigation (FBI)—Supplementary Homicide Report.

FIGURE 10. Percentage of homicides, by race* of victim and offender — United States, 1988†



*Race categories are black and white only.

†1988 FBI data do not include Florida or Kentucky.

Data source: Federal Bureau of Investigation (FBI) Supplementary Homicide Report.

Influenza – United States, 1989–90 and 1990–91 Seasons

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Summary

During the 1989–90 influenza season, 98% of all influenza viruses isolated in the United States and reported to CDC were influenza A. Almost all those that were antigenically characterized were similar to influenza A/Shanghai/11/87(H3N2), a component of the 1989–90 influenza vaccine. Regional and widespread influenza activity began to be reported in late December 1989, peaked in mid-January 1990, and declined rapidly through early April 1990. Most of the outbreaks reported to CDC were among nursing-home residents. Considerable influenza-associated mortality was reflected in the percentage of deaths due to pneumonia and influenza (P&I) reported through the CDC 121 Cities Surveillance System from early January through early April. More than 80% of all reported P&I deaths were among persons ≥65 years. In contrast to the predominance of influenza A during 1989–90, during the 1990–91 influenza season 86% of all influenza virus isolations reported were influenza B. Widespread influenza activity was reported from mid-January through April 1991, with regional activity extending into May. Outbreaks were reported primarily among schoolchildren, and no evidence of excess influenza-associated mortality was found. Almost all the influenza B isolates tested were related to influenza B/Yamagata/16/88, a component of the 1990–91 influenza vaccine, but were antigenically closer to B/Panama/45/90, a minor variant.

INTRODUCTION

Infections with influenza viruses remain an important cause of morbidity and mortality in the United States. During influenza epidemics, high attack rates of acute illness result in increased numbers of visits to physicians' offices, walk-in clinics, and emergency rooms and increased hospitalizations for lower-respiratory-tract complications (*1*).

Elderly persons and persons with underlying health problems are at increased risk for complications of influenza infection and are more likely than the general population to require hospitalization if infected (*2*). During major epidemics, hospitalizations for high-risk persons may increase two- to fivefold, depending on the age group. A

moderate epidemic of influenza A (H3N2) has been estimated to cost more than $300 million nationally in excess hospitalizations alone (*3*).

An increase in mortality from pneumonia and influenza (P&I), as well as cardiopulmonary and other chronic diseases that can be exacerbated by influenza infection, further indicates the impact of influenza epidemics. At least 20,000 excess deaths were associated with five of the U.S. influenza seasons in the period 1973–1981; more than 30,000 excess deaths occurred in each of three of these five seasons (*4*).

Every year from October through May, CDC's World Health Organization (WHO) Collaborating Center for Surveillance, Epidemiology and Control of Influenza monitors domestic influenza activity through four formal surveillance systems. Individually these systems are designed to systematically assess the impact of influenza on morbidity and mortality and to monitor the shifting predominance of influenza subtypes among circulating virus strains. This report summarizes CDC influenza surveillance information collected from all sources during the 1989–90 and 1990–91 influenza seasons in the United States.

METHODS

Sources for influenza surveillance during both 1989–90 and 1990–91 were similar to those in previous years:

1. *State and territorial reports.* Influenza activity, as assessed by the state and territorial epidemiologists, is reported on a weekly basis as widespread (outbreaks of influenza-like illness or culture-confirmed influenza in counties having a combined population of ≥50% of the state's population), regional (outbreaks of influenza-like illness or culture-confirmed influenza in counties having a combined population of <50% of the state's total population), sporadic (sporadically occurring cases of influenza-like illness or culture-confirmed influenza, with no outbreaks detected), or no activity.

2. *Sentinel physician surveillance network.* The number of physicians participating in this surveillance increased from 135 in 1989–90 to 139 in 1990–91. Weekly reports included the number of patients with influenza-like illness by age group per total number of patient visits per week, as well as the number of hospitalizations for influenza-like illness. A subgroup of 70 physicians in 1990–91 and 87 physicians in 1989–90 collected nasopharyngeal specimens from selected patients, which were processed for viral identification.

3. *World Health Organization (WHO) collaborating laboratories.* During 1989–90, 58 WHO collaborating laboratories (the majority from state or local health departments, with some university or hospital laboratories also participating) reported weekly the total number of specimens received for respiratory virus testing as well as the number and type of influenza viruses isolated. During 1990–91, the number of WHO collaborating laboratories increased to 64; of these, four also functioned as Health Care Financing Administration (HCFA) surveillance laboratories.*

Similar reports were received during 1990–91 from 12 additional laboratories that conducted influenza culture surveillance for HCFA demonstration project sites.

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*In 1988, Congress mandated grant funding for the Influenza Vaccine Demonstration Project with a goal of determining a) the cost-effectiveness of Medicare coverage of influenza vaccination and b) whether Medicare reimbursement and other measures to enhance vaccine delivery result in increased influenza vaccination levels among Medicare Part B beneficiaries (i.e., persons aged ≥65 years or persons of any age with a disability or who have end-stage renal disease).

4. *CDC 121 Cities Surveillance System.* Each week, the vital statistics offices of 121 cities reported the total number of death certificates filed due to all causes for that week and the percentage of those for which pneumonia was identified as the underlying cause of death or for which influenza was mentioned in any position. These data are graphed against a seasonal baseline calculated by using a robust regression procedure. An "epidemic threshold" for each season is 1.645 standard deviations above the seasonal baseline, which is calculated by using a periodic regression model applied to observed percentages since 1983; this measure provides an index for the severity of influenza activity (*4*).

RESULTS

1989–90

State and territorial epidemiologists

From September 23, when state epidemiologists in Nevada and Rhode Island first reported sporadic cases of influenza-like illness, to November 25, 1989, as many as 15 (27%) state and territorial epidemiologists throughout the country had reported sporadic levels of influenza-like illness. The first state health departments to report sustained regional influenza activity were in Montana during the week ending December 2 and in Massachusetts the following week. By December 16, six states reported widespread or regional influenza activity. The number of states reporting regional or widespread activity rapidly increased over the next 4 weeks. By January 27, 1990, 38 states (76%) in all areas of the United States reported either regional or widespread activity. Influenza activity declined sharply over the next 8 weeks. By March 10, all but four states (Iowa and Tennessee, widespread; Arizona and Maryland, regional) were reporting sporadic or no influenza activity. Iowa continued to report widespread activity through March 24, 1990; then Iowa and Arizona continued to report regional activity through April 7, when reports of sporadic influenza activity were received from 13 states in all regions of the country, plus Puerto Rico and the Virgin Islands. By April 28, only five states reported sporadic influenza activity (Figure 1).

Sentinel physicians surveillance network

The proportion of patient visits to sentinel physicians attributed to influenza-like illness first began to rise above a baseline of 2.5%–4% the week ending December 9 (week 49) and rose rapidly to a sustained peak of 8.1%–8.9% of all office visits during December 30, 1989–January 27, 1990 (weeks 52–4). The proportion of visits to sentinel physicians due to influenza-like illness fell rapidly during February and by March 17 (week 1) had returned to a baseline of <4%, where it remained throughout the duration of the surveillance season (Figure 2). Overall, 3.2% of patients who sought medical treatment for influenza-like illness were hospitalized. Reporting patterns were similar in all regions except the East North Central region (Figure 3), where a more sustained peak (>9% of all office visits) occurred from December 16, 1989, through February 10, 1990 (weeks 50–6). Nationally, hospitalizations peaked during January 6–20, 1991 (weeks 1–3), when 4.5%–6.1% of patient visits for influenza-like illness resulted in hospitalizations. Persons ≥65 years accounted for only 11% of patient visits for influenza-like illness, but 50% of all hospitalizations

related to influenza-like illnesses. Sentinel physicians submitted 318 specimens for respiratory virus testing; 113 (36%) were positive for influenza viruses, all of which were typed as influenza A.

WHO collaborating laboratories

The 58 WHO collaborating laboratories tested 30,060 specimens for isolation of respiratory viruses. Influenza virus isolates were identified and reported to CDC for 3,410 (11.3%) of these specimens; 2,678 (78.5%) were influenza A (H3N2), 37 (1.1%) were A (H1N1), and 682 (20%) were A (not subtyped). Thirteen (<1%) were influenza B (Figure 4).

In September 1989, the first influenza virus strain reported in the United States during the 1989–90 season (an A/Shanghai/11/87-like [H3N2] virus) was isolated from a Wisconsin student who became ill within 48 hours of returning from West Africa. Arizona and Montana first reported the isolation of influenza virus from persons whose illnesses were clearly acquired in the United States during the week ending November 8, 1989; both isolates were influenza A (H3N2). Isolation of influenza A (H3N2) peaked in late January and early February (Figure 5). Isolations of influenza A (H1N1) were clustered primarily during weeks 3–10 (January 14–March 10, 1990), and isolations of influenza B were sporadic from the beginning of January through the end of March. From all sources, influenza A (H3N2) viruses were reported from all 50 states, influenza A (H1N1) from 11 states, and influenza B from 14 states.

Almost all the influenza A (H3N2) viruses isolated during the 1989–90 season and characterized at CDC were antigenically related to A/England/427/88; most antigenically characterized influenza A (H1N1) isolates resembled A/Taiwan/1/86. Antigenically characterized influenza B isolates resembled B/Victoria/02/87 or B/Hong Kong/22/89.

The influenza reference viruses discussed here are reacted with serum specimens from ferrets infected with the reference viruses. The resulting hemagglutination titers are used to infer antigenic relationships between viruses (Table 1). Differences of fourfold in titer of a serum with two viruses are normally indicative of an experimentally significant variation between the viruses. In some cases, only asymmetric differences are seen when several variants are tested simultaneously. The reference virus A/England/427/88 is a minor antigenic variant of the reference virus A/Shanghai/11/87. Both A/Shanghai/11/87 (H3N2) and A/Taiwan/1/86 (H1N1) were components of the 1989–90 trivalent influenza vaccine. B/Hong Kong/22/89-like viruses are closely related to the reference virus B/Yamagata/16/88, which was the third component of the 1989-90 influenza vaccine.

121 Cities Surveillance System

The proportion of all deaths reported through the CDC 121 Cities Surveillance System that were attributed to P&I exceeded the epidemic threshold from week 1, ending January 6, 1990, through week 14, ending April 7, 1990, with a peak at 8.4% of all reported deaths during week 5, ending February 3, 1990 (Figure 6). Approximately 80% of the deaths from P&I reported during this period were among persons ≥65 years.

The first outbreaks of influenza-like illness reported to CDC occurred in elementary and secondary schools in Arizona and Ohio during November. Influenza A virus was isolated from students in both outbreaks. From December through mid-February, outbreaks were reported to CDC from 24 states, with 18 (75%) of those states

reporting outbreaks in nursing homes. Influenza A (not subtyped) or influenza A (H3N2) was associated with all reported outbreaks. No outbreak reports were received after mid-February.

1990–91

State and territorial epidemiologists

In mid-November 1990, state epidemiologists first reported sporadic influenza activity in the Mid-Atlantic and New England regions (Figure 3). By mid-January 1991, widespread influenza activity had been reported in New York; regional activity had been reported in six states east of the Mississippi River (Connecticut, Maryland, Massachusetts, New Hampshire, New Jersey, Rhode Island) and in Missouri and Nebraska. Influenza activity in the United States peaked from February 9 (week 6) through March 2 (week 9), when eight to 10 states (predominantly from the four central regions of the country) reported widespread activity each week. Smaller numbers of state epidemiologists, primarily in the Mountain and Pacific regions, continued to report widespread activity through the first week of April. Sporadic reports of regional influenza activity were received into May (Figure 1).

Sentinel physicians surveillance network

The proportion of patient visits to sentinel physicians attributed to influenza-like illness first began to rise above the baseline of 2.5%–3.5% in late November 1990 (week 47). Nationally, peak activity (>8.0% of office visits) occurred during the first 3 weeks of February 1991 (weeks 5–7) (Figure 2). Sentinel physicians in the East North Central, South Atlantic, and Mountain regions reported the highest proportion of overall office visits attributed to influenza-like illness for the season, ranging from 6.5% to 7.0% (Figure 3). Persons ≥65 years accounted for 10% of the patient visits for influenza-like illness but 39% of the hospitalizations due to influenza-like illness. Sentinel physicians submitted 221 specimens for respiratory virus testing; 58 (26%) of these were positive for influenza viruses. Of the positive specimens, 44 (76%) were type B and 14 (24%) were type A.

WHO collaborating laboratories

The 64 WHO collaborating laboratories and the 12 HCFA demonstration site surveillance laboratories tested 32,852 specimens for respiratory virus isolation. Influenza virus isolates were identified and reported to CDC for 3,199 (9.7%) of these specimens; 2,746 (85.8%) were influenza B, 181 (5.7%) were influenza A(H3N2), 122 (3.8%) were influenza A(H1N1), and 150 (4.7%) were influenza A (not subtyped) (Figure 4). Isolation of influenza B peaked in early to middle February (weeks 5–7) (Figure 5). In most of the 10 regions of the country, influenza B accounted for 88%–98% of reported isolations. In the West South Central, Mountain, and Pacific regions, the proportion of influenza B isolates ranged from 60% to 77% (Figure 3). Almost all the influenza B isolates tested were related to influenza B/Yamagata/16/88, which was included in the vaccine for the 1990–91 season, but the isolates were antigenically closer to B/Panama/45/90, a minor variant (Table 1).

Sporadic reports of influenza A were received throughout the season. A slight increase in influenza A isolations began in mid-February and persisted throughout the rest of the season, peaking in late March (week 13) (Figure 5). By June 1, 1991, influenza A viruses constituted 14% of the influenza isolates reported this season. One

hundred eighty-one (60%) of the 303 influenza A viruses reported with known subtype were A(H3N2) strains, and 122 (40%) were A(H1N1) strains. Most of the A(H3N2) strains characterized were antigenically distinct from the 1990–91 vaccine strain, A/Shanghai/16/89. Ninety-six percent were determined to be more closely related to the reference strain A/Beijing/353/89. Antiserum to A/Beijing/353/89 reacted well with other recently identified minor variants (Table 1). All A(H1N1) strains antigenically tested at CDC closely resembled A/Taiwan/1/86, the 1990–91 vaccine strain.

When reports from the 64 WHO collaborating laboratories alone are analyzed, 2,037 (7.9%) influenza isolates were reported from 25,614 specimens tested. Of these, 1,720 (84%) were influenza B, 140 (7%) were influenza A(H3N2), 68 (3%) were influenza A(H1N1), and 109 (5%) were influenza A (not subtyped). The temporal and geographic distribution of influenza types and subtypes did not differ significantly from that described by the combined reporting laboratories. The four WHO collaborating laboratories that also functioned as HCFA surveillance laboratories reported augmented influenza culture surveillance compared with reports prior to 1988.

All laboratory reports combined show that influenza B viruses were reported from 48 states and the District of Columbia, influenza A(H3N2) from 21 states and the District of Columbia, and influenza A(H1N1) from 19 states.

121 Cities Surveillance System

Throughout the 1990–91 season, the proportion of deaths associated with P&I reported through the CDC 121 Cities Surveillance System remained at levels consistent with a low mortality season (Figure 6). Eighty-one percent of the reported pneumonia and influenza deaths occurred among persons ≥65 years of age.

Outbreak reports

The first outbreak of influenza-like illness reported to CDC occurred in a New York State elementary school the first week of December. Influenza B was isolated from the index patient. All outbreaks reported before March 1991 were associated with influenza B, with illness occurring predominantly among school-age children. Increasing reports of influenza A isolates by the WHO collaborating laboratories beginning in late February were associated with a recrudescence of outbreak reports, primarily in nursing homes. The first culture-confirmed outbreak of influenza A(H3N2) occurred the third week of March in a military facility in Colorado. Culture-confirmed outbreaks of influenza A were reported through early May.

DISCUSSION

Two control measures are available in the United States that can reduce the mortality and morbidity associated with influenza: a) immunoprophylaxis with inactivated (killed-virus) vaccine and b) chemoprophylaxis or therapy with an influenza-specific antiviral drug (e.g., amantadine) (*5*). Long-term control of influenza by vaccination is complicated by the propensity of the virus for antigenic variation. Antigenic drift, the gradual introduction of small changes in the antigenic structure of the surface proteins, allows the virus to circumvent the existing immunity in a segment of the population on a year-to-year basis. Antigenic shift, the appearance of a virus with a major change in antigenic properties, occurs from time to time and results in the introduction of a virus with which a substantial proportion of the population may have no immunologic experience (*6*). Since the first isolations of an

influenza virus (later characterized as influenza A[H1N1]) in 1933, antigenic shifts have been recognized with the emergence of influenza A(H2N2) in 1957 and of influenza A(H3N2) in 1968. Since the reemergence of influenza A(H1N1) in 1977, influenza B, influenza A(H3N2), and influenza A(H1N1) viruses have continued to co-circulate in the population (*7*). Antigenic drift has occurred in viruses in all three groups, necessitating annual reformulation of the vaccine. To permit adequate time for the production, distribution, and administration of influenza vaccine, the vaccine composition each season is regularly decided during January–March of the previous season on the basis of worldwide surveillance of influenza strains. As a result, the congruence between virus antigens included in the vaccine and those present on circulating viruses varies from season to season (*8,9*).

The predominance of influenza A(H3N2) among influenza isolates during the 1989–90 epidemic (98% of all subtyped influenza isolates) exceeded that of any circulating strain during recent influenza seasons in the United States. The only comparable season during the past decade was 1984–85, when influenza A(H3N2) isolates accounted for 97.3% of total subtyped influenza isolates. Likewise, 1979–80 was the last season in which the predominance of influenza B among influenza isolates in the United States exceeded that of the 1990–91 season (86% of all isolates). Within the past decade, only the 1981–82 and 1985–86 seasons (when 75% and 76%, respectively, of all isolates were influenza B) approached the 1990–91 season in level of influenza B activity.

Comparison of influenza surveillance summaries from the 1989–90 and 1990–91 influenza seasons provides a classic contrast of the characteristic historic behavior of influenza A(H3N2) and influenza B in human populations. The 1989–90 influenza season was slightly shorter and more intense than the 1990–91 season. State and territorial epidemiologists reported a rapid onset of regional and widespread outbreaks of influenza-like illness in all areas of the country, beginning in early December and peaking in late January. The decline in evidence of influenza activity that began in February was virtually complete by April. Visits to sentinel physicians for influenza-like illness exceeded a baseline of 4% of office visits for only 14 weeks, from December 2, 1989, to March 10, 1990. Outbreaks of influenza A, reported from only two states during November, were reported from 24 states during December through February, predominantly in nursing homes. The number of influenza isolates, the percentage of patients with influenza-like illness seen by sentinel physicians, and the activity levels reported by state and territorial health departments did not indicate exceptionally high levels of influenza morbidity during the 1989–90 season. However, the proportion of all deaths reported to CDC through the 121 Cities Surveillance System exceeded the epidemic threshold for 12 weeks from January through late March. Greater than 80% of all reported deaths from P&I occurred among persons ≥65 years, reflecting the excess mortality in the elderly historically attributable to influenza A (H3N2).

In contrast, influenza activity in the United States during the 1990–91 season was temporally more diffuse and milder. Reports of widespread levels of influenza activity were received from state and territorial epidemiologists from mid-January through April, with regional activity continuing through May. The percentage of visits to sentinel physicians attributed to influenza-like illness was similar to that seen for the previous season, but exceeded the baseline (4%) for 20 weeks between late November and early April, with school-age children accounting for the highest morbidity.

Outbreaks, first reported in early December, continued through May. All outbreaks reported prior to March were due to influenza B in school-age children. Late spring reports of outbreaks in nursing homes accompanied an increase in reports of isolations of influenza A virus. Nevertheless, influenza-associated mortality was minimal, as evidenced by the low percentage of all reported deaths attributed to P&I.

The components of the trivalent 1989–90 influenza vaccine included A/Taiwan/1/86-like (H1N1), A/Shanghai/11/87-like (H3N2), and B/Yamagata/16/88-like virus antigens. The 1990–91 influenza vaccine was also trivalent and differed only in the A (H3N2) component (A/Shanghai/16/89-like [H3N2]). Both vaccines were well matched to the predominant influenza virus strains isolated in the United States during their respective seasons.

During the 1989–90 influenza season, when influenza A(H3N2) viruses predominated among circulating strains, excess influenza-associated mortality (primarily among the elderly) was clearly evidenced, and most reported outbreaks occurred in nursing homes. This pattern of activity is typical for influenza A(H3N2) viruses in human populations. In contrast, during the 1990–91 influenza season, when influenza B viruses predominated, no evidence of excess influenza-associated mortality was seen, outbreaks were reported primarily among school-age children, and late season reports of outbreaks in nursing homes were accompanied by an increase in influenza A virus isolations. Although this pattern of activity is typical for influenza B viruses, during the last influenza season when a comparable proportion (97%) of isolated viruses were influenza B (1979–80), excess influenza-related mortality did occur (*4*); influenza B has also been associated with outbreaks in nursing homes (*10*). Historically, however, influenza B viruses in human populations have been predominantly characterized by morbidity among school-age children and low influenza-associated mortality, as occurred during the 1990–91 influenza season.

References

1. Glezen WP, Couch RB. Interpandemic influenza in the Houston area, 1974–76. N Engl J Med 1978;298:587–92.
2. Glezen WP. Serious morbidity and mortality associated with influenza epidemics. Epidemiol Rev 1982;4:25–44.
3. Barker WH. Excess pneumonia and influenza associated hospitalizations during influenza epidemics in the U.S., 1970–1978. Am J Public Health 1986;76:761–5.
4. Lui KJ, Kendal AP. Impact of influenza epidemics on mortality in the United States from October 1972 to May 1985. Am J Public Health 1987;77:712–6.
5. CDC. Prevention and control of influenza: recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1992;41(No. RR-9).
6. Van Voris LP, Young JF, Bernstein JM, et al. Influenza viruses. In: Belshe RB, ed. Textbook of human virology. Littleton, Mass: PSG Publishing Company, Inc., 1984.
7. Nobel GR. Epidemiological and clinical aspects of influenza. In: Beare AS, ed. Basic and applied influenza research. Boca Raton: CRC Press, Inc., 1982.
8. Striver HG, Graves P, Eickhoff TC, et al. Efficacy of 'Hong Kong' vaccines in preventing 'England' variant influenza A in 1972. N Engl J Med 1973;289:1267–71.
9. Meiklejohn G, Eickhoff C, Graves P, et al. Antigenic drift and efficacy of influenza virus vaccines, 1976–77. J Infect Dis 1978;138:618–24.
10. CDC. Influenza—United States, worldwide. MMWR 1979;28:347–8.

TABLE 1. Hemagglutination titers of influenza viruses with serum specimens from infected ferrets*

Influenza A (H3N2)

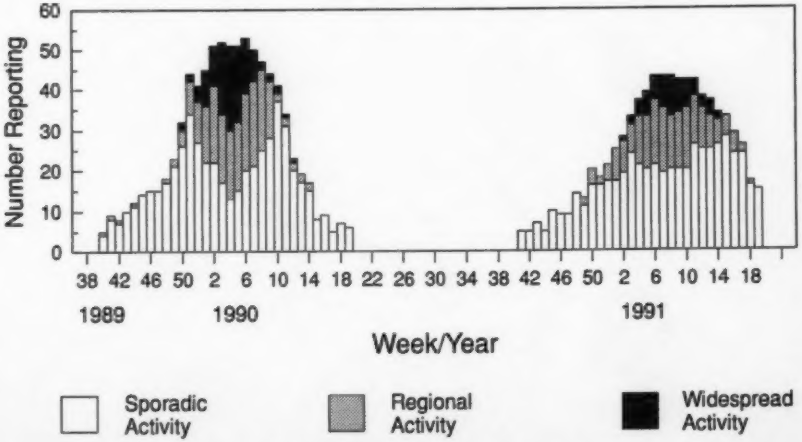
	Ferret antisera			
Reference antigen	**A/Shanghai/11/87**	**A/England/427/88**	**A/Shanghai/16/89**	**A/Beijing/353/89**
A/Shanghai/11/87	**1280**	320	320	640
A/England/427/88	640	**640**	320	320
A/Shanghai/16/89	80	160	**320**	40
A/Beijing/353/89	320	160	40	**320**

Influenza B

	Ferret antisera		
Reference antigen	**B/Victoria/2/87†**	**B/Yamagata/16/88**	**B/Panama/45/90**
B/Victoria/02/87	**320**	5	5
B/Yamagata/16/88	80	**640**	160
B/Panama/45/90	80	80	**160**

*Titers are used to infer antigenic relationships between viruses. Differences of fourfold in titer of a serum with two viruses are normally indicative of an experimentally significant variation between the viruses. In some cases, only asymmetric differences are seen when several variants are tested simultaneously.
†Sheep antisera.

FIGURE 1. Reports by state and territorial health departments for the 1989–1990 and 1990–1991 influenza seasons — United States



*Based on reports from the 50 states, New York City, Washington, D.C., Guam, Virgin Islands, and United States Pacific Jurisdiction.

FIGURE 2. Sentinel physician reports and percentage of office visits attributed to influenza-like illness — United States, 1989–1991

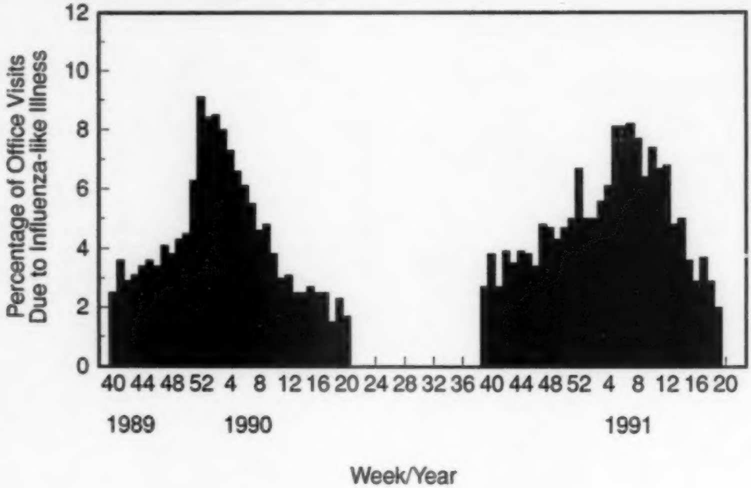


FIGURE 3. State and territorial health departments, by region

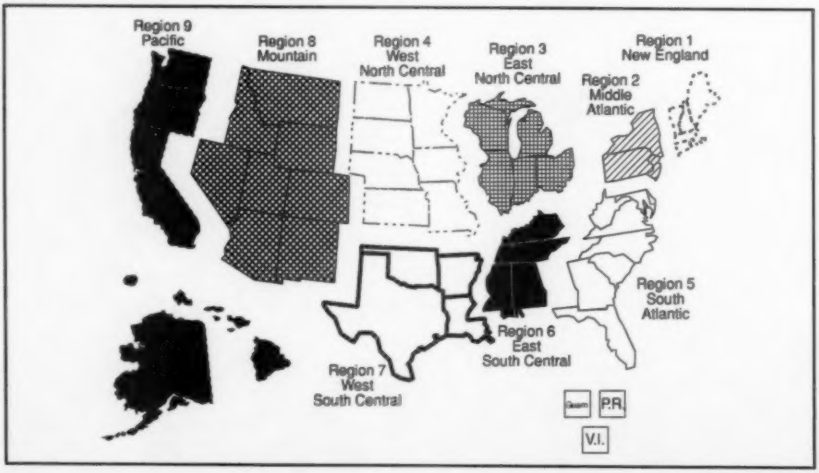
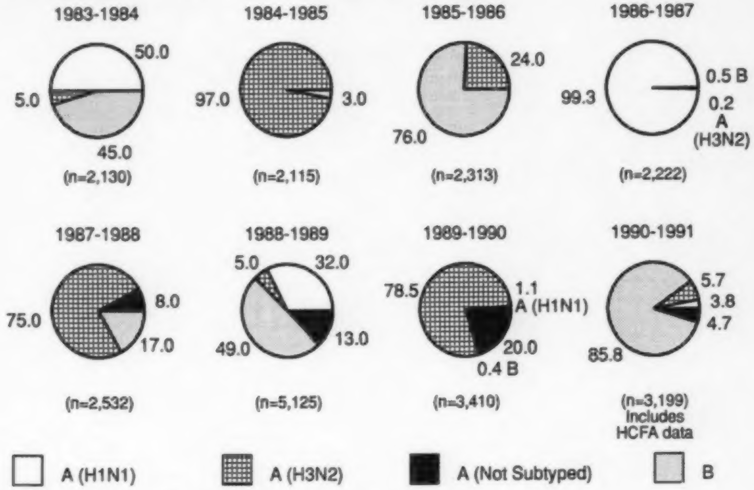


FIGURE 4. Influenza isolates reported by the World Health Organization and HCFA* collaborating laboratories — United States, 1983–1991



*HCFA = Health Care Financing Administration.

FIGURE 5. Influenza virus isolations reported by World Health Organization Collaborating Laboratories — United States, 1989–1991

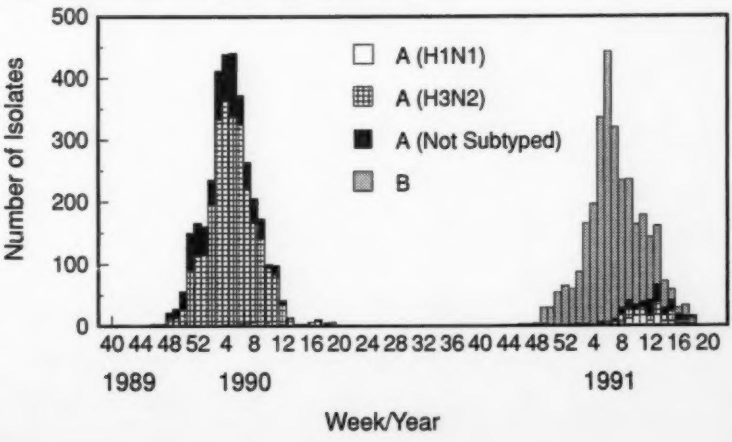
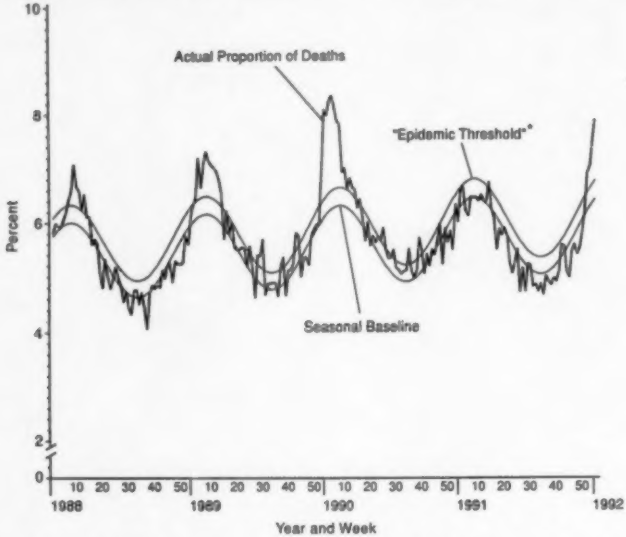


FIGURE 6. Percentage of all deaths attributable to pneumonia and influenza in 121 cities — United States, January 2, 1988–January 18, 1992



*The "epidemic threshold" for each season is 1.645 standard deviations above the seasonal baseline, calculated by using a periodic regression model applied to observed percentages since 1983. This baseline was calculated by using a robust regression procedure.

Laboratory-based Surveillance for Rotavirus United States, January 1989–May 1991

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Summary

Geographic and temporal trends of rotavirus detections in the United States for the period January 1989–May 1991 were determined by analyzing data reported monthly by 47 virology laboratories participating in the North American Rotavirus Surveillance System. Reports included complete information on the number of specimens tested, the number of test results positive for rotavirus, and the method used to detect rotavirus. Consistent trends in regional and geographic area were identified, with distinctly different peaks of rotavirus activity in the western and eastern states. Each year in the western states, rotavirus activity began in November and peaked in December–January, whereas in the eastern states activity began in January and peaked in February–March. These differences do not correlate with obvious trends in strain variation of rotavirus and remain unexplained. Unexpected reporting of summer rotavirus activity by some laboratories in 1989 was traced to the use of a single diagnostic kit and to two questionable laboratory practices: having more than six medical technologists perform the test and failure to use controls with the test. Laboratory-based surveillance of rotavirus activity has proven to be useful in identifying and correcting problems in laboratory methods for detecting rotavirus and will be a sensitive means for monitoring coverage of the rotavirus vaccine now being developed.

INTRODUCTION

Rotavirus infection, the most common cause of dehydrating diarrhea among children, results in an estimated 873,000 deaths per year worldwide. In the United States each year, rotavirus causes an estimated 75–125 deaths and 70,000 hospitalizations associated with severe diarrheal dehydration among children <5 years of age (*1–3*). Rotavirus infection may occur as early as 2–3 months after birth, and by the age of 4 years most children have been infected.

In 1989, on the basis of 5 years of retrospective data on the number of positive test results for rotavirus reported by virology laboratories in North America, CDC described unique geographic patterns of seasonal rotavirus activity (*4*). Activity began in Mexico and the southwestern United States in October and November, was greatest in the Midwest in January and February, and ended in the northeastern United States and Maritime Provinces of Canada in March–April.

In this report, data from laboratories providing the number of tests performed, the number of positive test results, and the detection methods used for the period

January 1989–May 1991 are presented. This summary describes the temporal and regional variations in the percentage of rotavirus-positive detections among stool specimens submitted to virology laboratories in North America and confirms the previously reported seasonal and geographic patterns of rotavirus diarrhea in the United States.

METHODS

Since January 1989, 98 virology laboratories in the United States, Canada, and Mexico have reported monthly by postcard to CDC the number of tests performed for rotavirus, the number of positive tests, and the detection method used (electron microscopy [EM], enzyme immunoassay [EIA], or latex agglutination [LA] test). For laboratories that were late, a reminder letter was generated monthly by computer and sent to the participant to request the missing data. CDC provided monthly computer-generated reports, including graphics, to the participating laboratories.

Surveillance Laboratories

Each of the laboratories in North America was assigned to one of nine regions (Bureau of the Census classification) (Table 1). Because the previous study (*4*) showed distinct differences between the seasonal patterns in eastern and western states, each laboratory was also classified as being in the "Western," "Central," or "Eastern" United States, according to Bureau of the Census criteria (Table 1). Of the 98 laboratories that have provided test results to CDC, 47 had provided CDC with complete data for the entire study period and were therefore included in the this analysis. These 47 centers, located in 37 states, included seven pediatric, 17 community, and 19 university hospital laboratories; three public health laboratories; and one commercial laboratory.

Because data from laboratories in Canada and Mexico were incomplete, they were not included in this analysis. In addition, reports from one laboratory in the central United States were excluded as outliers because its reported proportion (44%) of positive test results for the summers of 1989 and 1990 appeared to be substantially greater than those of other laboratories for the same periods.

Data Analysis

For this analysis, the monthly test results from all laboratories within a region were combined. The monthly percentage of rotavirus-positive specimens was calculated as the total number of positive test results divided by the total number of test results from all laboratories in that region. Similarly, the monthly percentage of positive test results for the three geographic areas was calculated from pooled data from all laboratories in that area.

RESULTS

Laboratory Methods, by Region

Of the 47 laboratories, 41 (87%) used EIA, four (9%) used LA, and two used EM to detect rotavirus (Table 2). Of the 41 laboratories using EIA, 21 used Pathfinder, Kallestad Laboratories; 16 used Rotaclone, Cambridge Bioscience Corp.; three used Rotazyme II, Abbott Lab.; and one used Testpack, Abbott Lab.

Seasonal Rotavirus Activity

For the 29–month period, the 47 laboratories tested 48,293 specimens for rotavirus; 10,208 (21.1%) were positive. During the winters of 1989–90 and 1990–91,

overall, rotavirus activity began in October and peaked during February in 1990 and March in 1991. For each of the two winter periods, the greatest number of rotavirus-positive detections was 791 (35%) in February 1990 and 958 (35%) in March 1991; the smallest number was 62 (6%) in June 1989 and 37 (4%) in September 1990.

Peak Rotavirus Activity, by Geographic Area

Each of the three geographic areas had a distinctly different pattern of seasonal rotavirus activity. The monthly rate of increase in the percentage of positive test results in the fall appears to be similar for all three areas, but the rate of decrease in the spring appears slower for the western area compared with rates for the eastern and central areas (Table 3, Figure 1).

The main difference in the rotavirus activity pattern, however, is in the months with the highest percentage of positive results. In the western area, the percentage positive began to increase in October and peaked in December, whereas in the eastern area it began in January and peaked in March. The central area had an intermediate pattern: the percentage of positive reports began increasing in December and peaked in January–February (Table 4, Figure 1). The differences in the seasonal and geographic patterns of rotavirus activity appear unlikely to be due to chance alone.

DISCUSSION

This 2–year prospective surveillance confirms the previous retrospective study (*4*) of regional and geographic area differences in peak rotavirus activity for the United States. The reason(s) for the geographic differences in seasonal rotavirus activity remain unclear. One hypothesis was that a single strain spreads from its point of introduction in the southwest toward the northeast. However, the tremendous diversity of strains in any given region suggests that, rather than a single introduction, the reservoir of rotavirus is more likely to be in the local environment (*5,6*). Why such local reservoirs result in the observed temporal and geographic trends remains an enigma.

An important issue in surveillance is that of missing data. Often laboratories report partial data for a month, or they may not perform tests for all months in the year. In this analysis, complete data (number tested and number positive) for the entire period were included. Because the criteria for inclusion were restricted, the seasonal and regional rotavirus activity curves on the graphs appeared smoother than those in the previous report (*4*).

Rotavirus activity is typically lowest during July and August. In 1989, a few laboratories reported higher than expected levels of rotavirus activity during these months. An investigation identified three factors that distinguished these laboratories from the rest: using one particular commercial assay, having more than six medical technologists perform the test, and failing to use simultaneous controls (*7*). The surveillance system provides a quality control mechanism to the laboratories by providing regular reports on their results and those of other laboratories around the country.

Data from the laboratory-based surveillance system are useful in identifying general patterns and trends. Test methods—EIA, LA, and EM, as well as name-brand test kits—have different sensitivities and specificities; for example, monoclonal antibody-based EIAs appear to be superior in specificity to the polyclonal antibody-based test for detection of rotavirus (*8*).

No demographic or clinical information was reported along with test results. We have not collected information on the patients' gender or race, other diagnoses, disease severity, or hospitalization status. A simple surveillance method with limited data was chosen to improve the timeliness and completeness of reporting.

Although rotavirus is not believed to be common in the elderly (>74 years of age), diarrhea is an underlying cause of death in this age group in the United States, particularly in the winter. From 1979 to 1987 in the United States, an annual average of 1,623 deaths among the elderly was caused by diarrheal disease (*9*), of which 51% occurred during the winter rotavirus peak. The seasonal patterns of diarrhea mortality among the elderly are different from those seen among children (*9*); the seasonal peak incidence of diarrhea mortality among the elderly precedes that among children by 1–2 months and does not demonstrate the unique geographic spread associated with rotavirus. Consequently, it is unlikely that the seasonal increase in deaths attributable to diarrhea among the elderly is due to rotavirus.

The only currently acceptable strategy for controlling morbidity and mortality due to rotavirus includes treatment with oral or intravenous rehydration solutions to prevent dehydration. To reduce mortality associated with rotavirus infection, health-care providers should initiate oral rehydration therapy for young children and the elderly (*10*). If proven effective in clinical trials, vaccines now being developed may play an important role in preventing rotavirus infection among children or modifying its clinical course (*11*). Information from the surveillance system already in place will improve our understanding of seasonal and geographic trends in rotavirus activity before the rotavirus vaccine is introduced, as well as provide evidence of the impact of the vaccine once it is developed and widely applied.

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References

1. Centers for Disease Control. Viral agents of gastroenteritis: public health importance and outbreak management. MMWR 1990;39(No. RR-5):1–24.
2. Glass RI, Lew JF, Gangarosa RE, et al. Estimates of morbidity and mortality rates for diarrheal diseases in American children. J Pediatr 1991;118:27–33.
3. Ho MS, Glass RI, Pinsky PF, et al. Diarrheal deaths in American children: are they preventable? JAMA 1988;260:3281–5.
4. LeBaron CW, Lew J, Glass RI, et al. Annual rotavirus epidemic patterns in North America. JAMA 1990;264:983–8.
5. Gouvea V, Ho MS, Glass RI, et al. Serotypes and electropherotypes of human rotavirus in the USA: 1987-1989. J Infect Dis 1990;162:362–7.
6. Matson DO, Estes MK, Burns JW, et al. Serotype variation of human group A rotaviruses in two regions of the USA. J Infect Dis 1990;162:605–14.
7. LeBaron CW, Allen JK, Hebert M, et al. Outbreaks of summer rotavirus linked to laboratory practices. Pediatr Infect Dis J 1992 (in press).
8. Dennehy PH, Gauntlett DR, Tente WE. Comparison of nine commercial immunoassays for the detection of rotavirus in fecal specimens. J Clin Microbiol 1988;26:1631–4.
9. Lew JF, Glass RI, Gangarosa RE, et al. Diarrheal deaths in the United States, 1979 through 1987. JAMA 1991;265:3280–4.
10. Avery ME, Snyder JD. Oral therapy for acute diarrhea. N Engl J Med. 1990;323:891–4.
11. Ing DJ, Glass RI, Woods PA, et al. Immunogenicity of tetravalent rhesus rotavirus vaccine administered with buffer and oral polio vaccine. Am J Dis Child 1991;145:892–7.

TABLE 1. States with laboratories participating in rotavirus surveillance network, by region and area, United States, 1989–1991*

Western area		
Mountain	Pacific	West South Central
Nevada (1)-†	Washington (1)	Oklahoma (1)
Montana (1)	Oregon (1)	Arkansas (1)
Idaho (0)	California (2)	Texas (3)
Utah (0)	Alaska (1)	Louisiana (1)
Arizona (1)	Hawaii (0)	
Wyoming (0)		
Colorado (1)		
New Mexico (0)		
Central area		
West North Central	East North Central	East South Central
N. Dakota (1)	Wisconsin (1)	Kentucky (1)
S. Dakota (1)	Illinois (2)	Tennessee (1)
Nebraska (0)	Michigan (1)	Mississippi (0)
Minnesota (2)		Alabama (0)
Iowa (0)		
Missouri (2)		
Eastern area		
New England	Mid-Atlantic	South Atlantic
Maine (1)	New York (2)	W. Virginia (0)
Vermont (0)	Pennsylvania (1)	Washington (1)
New Hampshire (1)	New Jersey (2)	Virginia (1)
Massachusett (3)	Indiana (1)	Delaware (1)
Connecticut (1)	Ohio (1)	Maryland (0)
Rhode Island (1)		N. Carolina (1)
		S. Carolina (2)
		Georgia (0)
		Florida (0)

*Based on Bureau of the Census criteria.
†Number of laboratories included in analysis appear in parentheses.

TABLE 2. Methods used by rotavirus surveillance laboratories for testing fecal specimens, by region and geographic area

Geographic area and region	Method*								
	KAL	RC	MER	RZII	TP	EM	IM	R-STAT	Total
Western area									
Mountain	2	–	–	–	–	1	1	–	4
Pacific	1	1	–	2	–	–	–	1	5
West South Central	5	1	–	–	–	–	–	–	6
Central area									
West North Central	3	1	–	–	1	–	–	–	5
East North Central	4	2	–	–	–	–	–	–	6
East South Central	–	2	–	–	–	–	–	–	2
Eastern area									
New England	3	5	–	–	–	–	–	–	8
Mid-Atlantic	2	1	1	1	–	–	–	–	5
South Atlantic	1	3	1	–	–	1	–	–	6
Total	**21**	**16**	**2**	**3**	**1**	**2**	**1**	**1**	**47**

*KAL, Pathfinder, Kallestad; RC, Rotaclone, Cambridge Bioscience Corp.; MER, Meritec-Rotavirus, Meridian Diagnostics, Inc.; RZII, Rotazyme II, Abbott Lab.; TP, Testpack, Abbott Lab.; EM, Electron Microscopy; IM, ImmunoSCAN, MicroScan; R-STAT, Isolab Inc.

TABLE 3. Seasonal pattern of rotavirus activity by geographic area and regions, June 1990–May 1991

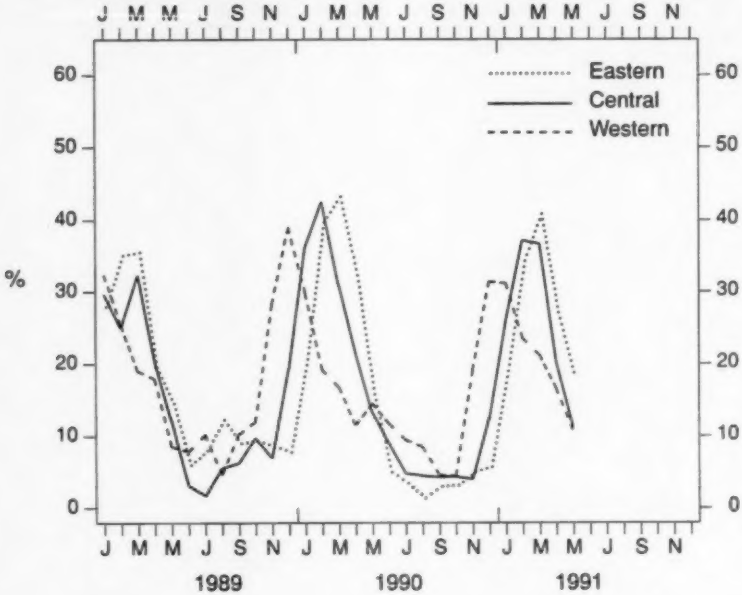
	Geographic area/Census region												
	Western area				Central area				Eastern area				
Month/ year	Mountain (4*)	Pacific (5)	W. S. Central (6)	Subtotal (15)	W. N. Central (5)	E. N. Central (6)	E. S. Central (2)	Subtotal (13)	New England (8)	Mid-Atlantic (5)	South Atlantic (6)	Subtotal (19)	Total (47)
Jun 90	12[†]/86[§] 14[¶]	5/106 5	32/228 14	49/420 12	12/105 11	23/218 11	3/102 3	38/425 9	6/137 4	12/143 8	1/110 1	19/390 5	106/1235 9
Jul 90	3/54 6	3/200 2	51/252 20	57/506 11	14/124 11	4/192 2	0/61 0	18/377 5	7/129 5	5/143 3	0/96 0	12/368 3	87/1251 7
Aug 90	3/62 5	3/168 2	31/207 15	37/437 8	10/124 8	7/188 4	0/70 0	17/382 4	2/107 2	2/135 1	0/91 0	4/333 1	58/1152 5
Sep 90	2/61 3	2/136 1	10/108 9	14/305 5	5/110 5	8/164 5	2/77 3	15/351 4	5/88 6	3/101 3	0/92 0	8/281 3	37/937 4
Oct 90	4/69 6	2/123 2	13/229 6	19/421 5	13/147 9	8/232 3	0/102 0	21/481 4	1/99 1	8/103 8	1/134 1	10/336 3	50/1238 4
Nov 90	17/98 17	39/198 20	52/271 19	108/567 19	6/145 4	14/221 6	0/129 0	20/495 4	6/145 4	10/134 7	3/111 3	19/390 5	147/1452 10
Dec 90	23/85 27	89/289 31	97/292 33	209/666 31	12/187 6	59/322 18	3/86 3	74/595 12	7/157 4	11/105 10	6/170 4	24/432 6	307/1693 18
Jan 91	27/98 28	72/315 23	145/372 39	244/785 31	42/178 24	121/441 27	47/191 25	210/810 26	7/159 4	54/228 24	61/262 23	122/649 19	576/2244 26
Feb 91	21/66 32	44/314 14	105/346 30	170/726 23	94/252 37	188/509 37	80/215 37	362/976 37	64/253 25	103/269 38	158/431 37	325/953 34	857/2655 32
Mar 91	3/51 6	38/197 19	79/320 25	120/568 21	115/274 42	150/450 33	85/232 37	350/956 37	140/381 37	165/342 48	183/476 38	488/1199 41	958/2723 35
Apr 91	12/70 17	40/215 19	29/206 14	81/491 16	39/179 22	60/294 20	22/128 17	121/601 20	114/361 32	48/203 24	55/243 23	217/807 27	419/1899 22
May 91	11/67 16	12/154 8	18/160 11	41/381 11	21/168 13	26/219 12	8/101 8	55/488 11	46/186 25	18/132 14	18/145 12	82/463 18	178/1332 13
Total	**138/867 16**	**349/2415 14**	**662/2991 22**	**1149/6273 18**	**383/1993 19**	**668/3450 19**	**250/1494 17**	**1301/6937 19**	**405/2202 18**	**439/2038 22**	**486/2361 21**	**1330/6601 20**	**3780/19811 19**

*Number of laboratories reporting.
[†]Number of tests positive for rotavirus.
[§]Number of specimens tested.
[¶]Percent positive.

TABLE 4. Percentage of positive test results, by geographic area, United States, January 1989–May 1991

Year/month	Western area	Central area	Eastern area
1989			
Jan	32.4	29.6	27.8
Feb	25.4	25.0	35.0
Mar	19.0	32.3	35.4
Apr	17.9	20.3	19.4
May	8.5	12.1	14.4
Jun	7.9	3.1	5.8
Jul	10.2	1.7	7.8
Aug	4.6	5.6	12.1
Sep	10.3	6.3	8.9
Oct	12.0	9.7	9.2
Nov	28.2	7.0	8.5
Dec	39.0	19.3	7.7
1990			
Jan	29.8	36.2	20.5
Feb	19.1	42.5	39.6
Mar	16.7	31.0	43.1
Apr	11.5	21.5	32.1
May	14.5	13.3	15.6
Jun	11.7	8.9	4.9
Jul	11.3	4.8	3.3
Aug	8.5	4.5	1.2
Sep	4.6	4.3	2.9
Oct	4.5	4.4	3.0
Nov	19.1	4.0	4.9
Dec	31.4	12.4	5.6
1991			
Jan	31.1	25.9	18.8
Feb	23.4	37.1	34.1
Mar	21.1	36.6	40.7
Apr	16.5	20.1	26.9
May	10.8	11.3	17.7

FIGURE 1. Percentage of positive test results for rotavirus, by geographic area, United States, January 1989–May 1991



Chancroid in the United States, 1981–1990: Evidence for Underreporting of Cases

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Summary

Chancroid, a bacterial sexually transmitted disease (STD) characterized by genital ulceration, has reemerged in the United States during the last decade. From 1950 to 1980, cases were infrequently reported. After an epidemic in California in 1981, however, the numbers of cases increased, peaking in 1987 at 5,035. Despite a subsequent decline in numbers of reported cases to 4,223 in 1990, new areas continue to report outbreaks. Interpreting chancroid surveillance data is difficult because confirmatory culture media are not commercially available. In addition, states may not require that unconfirmed or even confirmed cases be reported. To determine if chancroid is more widely distributed than surveillance figures indicate, CDC contacted STD clinics in 115 health departments, located in 32 states, the District of Columbia, and Puerto Rico—areas chosen because they had reported five or more cases of chancroid in any single year during 1986–1990—to determine if cases might be occurring but not reported. Only 16 of the 115 clinics had culture media available for Haemophilus ducreyi, *and only nine had laboratory facilities complete enough to definitively diagnose chancroid, syphilis, or genital herpes, the most common STDs characterized by genital ulcers. Five or more clinically likely cases occurring in 1990 were identified in 24 states, seven more than surveillance figures indicated. Surveillance can be improved if a) states utilize the definitions for chancroid cases adopted for use in 1990 and b) microbiology laboratories utilize enhanced diagnostic methods.*

INTRODUCTION

Chancroid cases were commonly reported early this century, peaked at 9,515 cases in 1947, and declined after the Korean War. An annual mean of 878 cases of chancroid was reported during the decade 1971–1980, but numbers of cases began to increase in 1981, when culture-confirmed outbreaks occurred in Orange County, California, (1981) and Orange County, Florida (1984) (Figure 1) (*1*). Other outbreaks have been concentrated in large cities on the Atlantic and Pacific coasts and in the South.

The initial California outbreak was linked to foreign workers lacking U.S. residency documents. These workers live in crowded conditions and have sexual contact with prostitutes, who spread the disease. Chancroid now occurs more widely among the American population, but prostitution remains important in transmission of *Haemophilus ducreyi* , the etiologic agent of chancroid, with some outbreaks linked to the exchange of sex for drugs (*1*). Most chancroid patients are heterosexual men (*1*).

In 1987, five states (New York, California, Texas, Florida, and Georgia) reported 93% of the 5,035 cases reported to CDC. In 1990, those same five states reported 88%

of the 4,223 cases reported; chancroid had also become established in North Carolina, Louisiana, and Massachusetts. Texas and New York reported 68% of all chancroid cases in 1990, but with different trends; during 1989–1990, the numbers of cases from Texas increased from 619 to 1,303 (a 111% increase), while the number of cases from New York declined from 2,305 to 1,596 (a 31% decrease).

Diagnosis and reporting of chancroid present a dilemma. Precise diagnosis requires culture of *H. ducreyi*, but culture media are not commercially available and, therefore, not widely used. Since 1987, at least seven outbreaks of suspected chancroid cases in Maryland, Ohio, Alabama, Tennessee, Georgia, Mississippi, and the District of Columbia were not reported because the clinical suspicion was not confirmed by culture. Many states have reported only culture-confirmed chancroid cases, in part because a standard definition for a clinically compatible case was not implemented until 1990 (*2*).

A clinical diagnosis of chancroid should be used if culture media for *H. ducreyi* are not available, but should be based on both physical findings and the laboratory exclusion of other sexually transmitted diseases (STDs) characterized by genital ulcers—syphilis and genital herpes. However, laboratory tests for syphilis and genital herpes sometimes are not performed, making even clinical diagnosis unreliable.

METHODS

Because of a suspicion that chancroid is more widely distributed than formal surveillance data indicate, CDC undertook a telephone survey in the fall of 1990, contacting STD clinics operated by 115 local health departments. Our goal was to determine whether chancroid cases were occurring but were not being reported. The clinics were located in states with cities of >200,000 population or in states that had reported five or more cases of chancroid in any single year from 1986 to 1990. A questionnaire was administered by telephone to either the medical director or STD program manager to determine a) whether confirmed or probable chancroid cases had occurred in 1990; b) the laboratory capacity to diagnose chancroid, syphilis, and genital herpes; and c) what control efforts were utilized for suspected chancroid cases. Because no definition for probable cases had existed before 1990, we defined a probable case of chancroid as an illness characterized by the following: one or more painful genital ulcers and inguinal adenopathy; clinical appearance not resembling genital herpes; and negative tests for syphilis (darkfield and/or serologic).

RESULTS

The 115 STD clinics, each operated by a city or county health department, that we surveyed were located in 32 states, Puerto Rico, and the District of Columbia; 74 (64%) reported confirmed/probable cases in 1990. Only 9 (8%) of the 115 clinics had complete laboratory evaluation available to determine whether patients' genital ulcers were chancroid (culture for *H. ducreyi*), syphilis (darkfield microscopy and serologic tests), or genital herpes (herpes simplex virus culture or Tzanck smear). Sixteen (14%) of the 115 clinics could perform culture for *H. ducreyi*. Only 48 (42%) clinics could make a clinical diagnosis of chancroid by first excluding syphilis (darkfield microscopy and serologic testing) and genital herpes (herpes simplex virus culture or Tzanck smear). Ninety-one (79%) clinics could exclude syphilis through use of both darkfield microscopy and serologic testing; the remaining 24 clinics lacked darkfield microscopy.

Of the 74 clinics reporting chancroid cases in 1990, staff conducted interviews to elicit names of sex partners in 21 (28%), and 15 (20%) attempted active partner notification. In the remaining clinics, patient referral of sex partners was encouraged.

DISCUSSION

Precise reasons for the decline in numbers of reported cases of chancroid since 1987 are unclear. However, problems with the accurate diagnosis of chancroid and subsequent reporting of possible cases complicates interpretation of surveillance data.

Our results indicate that chancroid is more geographically widespread than the distribution of reported cases would suggest and that the numbers of reported cases may not accurately reflect the actual numbers. Only 17 states officially reported five or more cases to CDC in 1990, but our survey found cases in 24 states and Puerto Rico.

Chancroid is underreported for two reasons. First, as this survey indicates, availability of culture media for *H. ducreyi* is limited. Even in laboratories with media and experience in culturing *H. ducreyi*, however, microbiologists are able to isolate *H. ducreyi* in only about 60%–70% of patients with clinically compatible cases (*3*). Second, because no uniform reporting definition of probable cases existed until recently, some states reported only culture-confirmed cases; hence, even potentially large numbers of clinically compatible cases were not reported.

To enhance surveillance and control of chancroid in the United States, several problems with diagnosis and reporting should be addressed. First, improved methods of laboratory testing must be developed. Although culture is insensitive and requires media not commercially available, it remains the most specific way to diagnose chancroid and should be more widely used. Second, the newly adopted definition for probable cases should be used by STD programs for reporting purposes (Table 1) (*2*). In the United States, a patient seeking medical attention for a genital ulcer most likely has chancroid, syphilis, or genital herpes (*4*). When *H. ducreyi* culture is not available, practitioners should be encouraged to make clinical diagnoses of chancroid after syphilis and genital herpes have been excluded with laboratory tests.

Finally, when confirmed or probable chancroid cases are detected, sex partners should be identified, evaluated, and treated promptly. Partner notification may be done by patients (patient referral) or providers (provider referral). Our study indicates only one in five STD clinics utilize provider referral for suspected chancroid cases and, of the states surveyed, only California mandates provider referral by the health department for chancroid cases. However, partner notification has proved to be one effective tool in controlling outbreaks (*5*); other tools include intensive case finding and investigation, especially among prostitutes, and treatment of all sexual contacts.

In studies done in Africa (*6*) and the United States (*7,8*), chancroid—and other diseases characterized by genital ulcers—appears to facilitate transmission of, and be highly associated with, the human immunodeficiency virus (HIV). The association of chancroid with HIV transmission is an additional reason to promote chancroid control efforts. All chancroid patients should be encouraged to have HIV counseling and testing, to reduce their behavioral risk factors, and to be evaluated for other STDs.

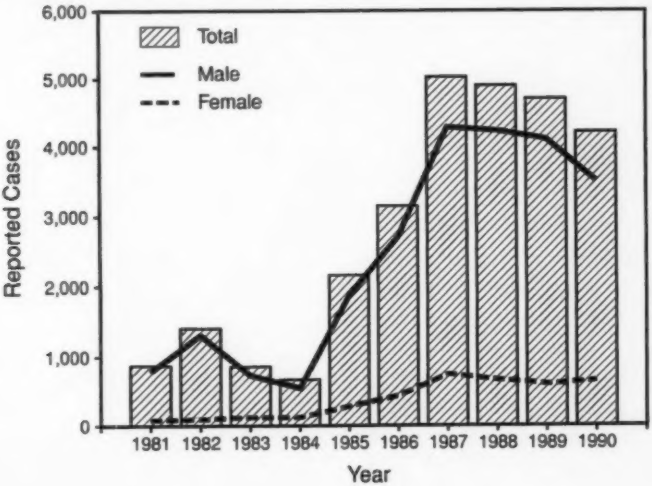
References

1. Schmid GP, Sanders LL, Blount JH, et al. Chancroid in the United States: reestablishment of an old disease. JAMA 1987;258:3265–8.
2. CDC. Case definitions for public health surveillance. MMWR 1990;39(No. RR-13):8–9.
3. Morse SA. Chancroid and *Haemophilus ducreyi*. Clin Microbiol Rev 1989;2:137–57.
4. Schmid GP. Approach to the patient with genital ulcer disease. Med Clin North Am 1990;74:1559–72.
5. Jessamine PG, Brunham RC. Rapid control of a chancroid outbreak: implications for Canada. Can Med Assoc J 1990;142:1081–5.
6. Jessamine PG, Ronald AR. Chancroid and the role of genital ulcer disease in the spread of human retroviruses. Med Clin North Am 1990;74:1417–31.
7. McLaughlin M, Wilkes M, Blum S, et al. Risks associated with acquiring chancroid genital ulcerative disease and HIV infection: a case control study. In: Programs and Abstracts of the Fifth International Conference on AIDS, Montreal, Canada, June 1989.
8. Telzak EM, Chiasson M, Bevier P, et al. Chancroid and the high risk of HIV seroconversion in male heterosexuals in New York City. In: Programs and Abstracts of the Seventh International Conference on AIDS, Florence, Italy, June 1991.

TABLE 1. Classification for cases of chancroid: laboratory criteria for diagnosis

Isolation of *Haemophilus ducreyi* from a clinical specimen
Case classification
Probable: a clinically compatible case with one or more painful genital ulcers and both
a) no evidence of *Treponema pallidum* infection by darkfield examination of ulcer exudate or by a serologic test for syphilis performed at least 7 days after onset of ulcers
and
b) the clinical presentation of the ulcers is not typical of disease caused by herpes simplex virus (HSV) or HSV culture is negative
Confirmed: a case that is laboratory confirmed.

FIGURE 1. Chancroid cases, by gender — United States, 1981–1990



State and Territorial Epidemiologists and Laboratory Directors

State and Territorial Epidemiologists and Laboratory Directors are gratefully acknowledged for their contributions to this report. The epidemiologists listed below were in the positions shown as of May 22, 1992, and the laboratory directors listed below were in the positions shown as of April 2, 1991.

State/Territory	Epidemiologist	Laboratory Director
Alabama	Charles H. Woernle, MD, MPH	William J. Callan, PhD
Alaska	John P. Middaugh, MD	Katherine A. Kelley, DrPH
Arizona	Steven J. Englender, MD, MPH	Thomas S. Davis
Arkansas	Thomas C. McChesney, DVM	Robert L. Horn
California	George W. Rutherford, MD	Michael G. Volz, PhD
Colorado	Richard E. Hoffman, MD, MPH	Ronald L. Cada, DrPH
Connecticut	James L. Hadler, MD, MPH	Sanders Hawkins, PhD
Delaware	Paul R. Silverman, DrPH	Mahadeo P. Verma, PhD
District of Columbia	Martin E. Levy, MD, MPH	James B. Thomas, ScD
Florida	Richard S. Hopkins, MD, MSPH	E. Charles Hartwig, ScD
Georgia	Joseph A. Wilber, MD	Elizabeth A. Franko
Hawaii	Eugene Pon, MD, MPH	Vernon K. Miyamoto, PhD
Idaho	Fritz R. Dixon, MD	Richard Hudson, PhD
Illinois	Byron J. Francis, MD, MPH	David F. Carpenter, PhD
Indiana	Mary Lou Fleissner, DrPH	Gregory V. Hayes, DrPH
Iowa	Laverne A. Wintermeyer, MD	W. J. Hausler, Jr, PhD
Kansas	Andrew R. Pelletier, MD	Roger H. Carlson, PhD
Kentucky	Reginald Finger, MD, MPH	Thomas E. Maxson, DrPH
Louisiana	Louise McFarland, DrPH	Henry B. Bradford, Jr, PhD
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